Journal of Neurosurgery

MARQUETTE UNIVERSITY
1955
Medical-Dental Library

JANUARY 1955

VOLUME XII JANUARY 1955 NUMBER 1

JOURNAL OF NEUROSURGERY

[*J. Neurosurg.*]

MANAGING EDITOR

LOUISE EISENHARDT, 333 Cedar Street, New Haven 11, Conn.

EDITORIAL BOARD

WINCHELL McK. CRAIG, Rochester, Minn., *Chairman*

PAUL C. BUCY, Chicago
FRANC D. INGRAHAM, Boston
BRONSON S. RAY, New York
BARNES WOODHALL, Durham

ADVISORY BOARD

A. ASENJO, Santiago de Chile
GILBERT HORRAX, Boston
GEOFFREY JEFFERSON, Manchester, Eng.
KENNETH G. McKENZIE, Toronto
W. JASON MIXTER, Boston
HOWARD C. NAFFZIGER, San Francisco
HERBERT OLIVECRONA, Stockholm
WILDER PENFIELD, MONTREAL
D. PETIT-DUTAILLIS, Paris
ERNEST SACHS, New Haven
R. GLEN SPURLING, Louisville

PUBLISHED BI-MONTHLY

FOR THE HARVEY CUSHING SOCIETY BY

CHARLES C THOMAS, Publisher

301-327 EAST LAWRENCE AVENUE, SPRINGFIELD, ILLINOIS, U.S.A

Copyright, 1955, by Charles C Thomas, Publisher. Subscription rates: $11.00 a year in the United States, Pan-American Union, and Spain; $11.50 a year in Canada; $12.00 a year in other countries; single copies, $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available.

Distributed in the British Commonwealth of Nations by Blackwell Scientific Publications, Ltd., 24–25 Broad Street, Oxford, England. Copyrighted in the British Commonwealth by Blackwell Scientific Publications, Oxford. No part may be duplicated or reproduced without permission of publishers. Subscription price £4.7.6.

Entered as second-class matter March 12, 1944 at the post office at Springfield, Illinois, under the act of March 3, 1879. Additional Entry at the post office at Menasha, Wisconsin.

PRINTED IN U.S.A.

Stop bleeding

from broad capillary bed . . .

Adrenosem (adrenochrome semicarbazone sodium salicylate), a synthetic complex, is a unique systemic hemostat, administered orally or parenterally.

It will not raise blood pressure nor affect cardiac rate or volume. It does not affect blood components associated with clot formation.

Adrenosem has such a high index of therapeutic safety that there are no contraindications in the recommended dose. Case histories attest its value as a hemostat in surgery and in pathologies characterized by hemorrhagic tendencies.*

*Sherber, D., Am. J. Surg., Sept. 1953

U. S. Pat. 2,581,850—1952

Adrenosem

indicated in

Idiopathic purpura
Prostatic surgery
Retinal Hemorrhage
Nasopharynx surgery
Familial telangiectasia
Postoperative
Bladder surgery
Tonsillectomy
Hemoptysis
Epistaxis

send for professional literature

Massengill BRISTOL, TENN.

Fortschritte der Hals-Nasen-Ohrenheilkunde

Advances in Oto-Rhino-Laryngology

Progrès en Oto-Rhino-Laryngologie

herausgegeben von/edited by/dirigé par

Professor L. Rüedi, M.D., Zurich, Switzerland

Advances in Oto-Rhino-Laryngology pursue the aim of giving a complete and at the same time critical survey on the laryngologic literature of the world. They will be specially welcome to laryngologists unable to read the innumerable original papers continuously coming forth. Each paper published in the "*Advances in Oto-Rhino-Laryngology*" contains an extensive bibliography that will be of high value to every research worker in the field.

Vol. 1

VIII + 263 p., 49 fig., 1953, Swiss frs. 36.00/ $8.80

DER MENSCHLICHE VESTIBULARAPPARAT

L. B. W. JONGKEES, Amsterdam: **Über die Untersuchungsmethoden des Gleichgewichtsorgans.**

M. ARSLAN, Padua: **Über die Methodik der Vestibularisuntersuchungen und Vorschlag zur Standardisierung.**

E. WODAK, Tel-Aviv: **Physio-Pathologische Probleme des menschlichen Vestibularapparates.**

Vol. 2

VI + 274 p., 40 fig., 1955, Swiss frs. 36.00/ $8.80

FRANZ ALTMANN, New York: **Morbus Ménière**

FRANZ ALTMANN, New York: **Entzündliche und degenerative Erkrankungen des peripheren Cochlear- und Vestibular-Neurons**

KURT GRAF, Zürich: **Die Kleinhirnbrückenwinkelgeschwülste**

Volume 1:

Archives of Otolaryngology (October 1953): "This volume deals with the vestibular apparatus. Jongkees discusses many of the newer theories of the vestibular function and the physiology of the vestibular organ. He also outlines the significance of both normal and pathologic reactions to the various forms of stimulation. Arslan presents a chapter on methods of testing the vestibular apparatus. Wodak discusses the physiopathologic problems and presents many case reports and the interpretation of abnormal findings from the standpoint of differential diagnosis of both peripheral and central lesions, with a considerable discussion of Ménière's Syndrome. This book, replete with many references, is unquestionably a very comprehensive evaluation of our present knowledge of the vestibular organ."

BASEL 11 (Switzerland) S. KARGER NEW YORK

For U.S.A.: Albert J. Phiebig, P.O. Box 352, White Plains, N.Y.

Cranioplastic

RESINOUS MATERIAL FOR REPAIRING CRANIAL DEFECTS

A method of rapidly repairing cranial defects using a resin material containing Methyl-methacrylate which is molded into the cranial defect at the time of the original operation. This highly refined plastic has been used since 1947 and to date it has not been necessary to remove a single plate.

N-196 CRANIOPLASTIC KIT

Contains 3 packets of powder (sterile), 3 ampules of liquid (sterile) and 3 matrix bags which is sufficient material for the repair of three ordinary cranial defects.

N-197 SPENCE CRANIOPLASTIC ROLLER

A stainless steel roller which is of great help in rolling the plastic mass to the desired thickness before fitting to the cranial defect.

N-198 CARBORUNDUM WHEEL

Used with bone saw or drill to bevel edges and smooth rough spots. Can be autoclaved.

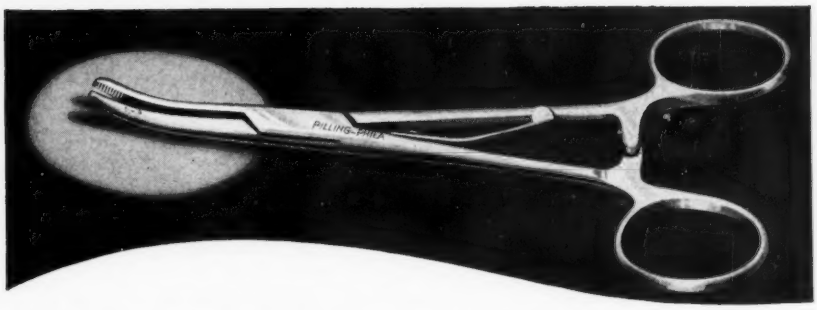
Ref.: Spence, William T.: Form Fitting Plastic Cranioplasty. Journal of Neurosurgery. May, 1954.

ESTABLISHED 1838

Codman & Shurtleff, Inc.

MAKERS OF SURGEONS' INSTRUMENTS

104 BROOKLINE AVENUE BOSTON 15, MASSACHUSETTS



Uses "U" clips

or "V" clips

CLIP APPLYING FORCEPS *as modified by John H. Drew, M.D., Boston*

Clips will not stick or jam

In actual operating room use, Drew Forceps

1. entirely eliminate the problem of clips jamming or catching in the jaws of the forceps
2. hold clips more securely—they are not dislodged during transfer from nurse to surgeon
3. are easier to use—with Drew forceps the nurse picks clips off the McKenzie magazine with much less effort or trouble than with the standard forceps.

Write for complete listing of clip applying forceps with Drew jaws.

Ref.: Drew, J.H.: J. Neurosurg. 10:439 (July) 1953.

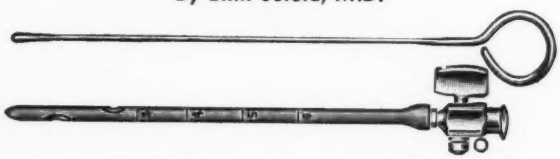
THE AUTHENTIC INSTRUMENT IS AVAILABLE ONLY FROM PILLING . . . *ORDER DIRECT:*

GEORGE P. **Pilling** & SON CO.

3451 WALNUT STREET . . . . . PHILADELPHIA

New Non-Rigid Ventricular Cannula

By Emil Seletz, M.D.



This new, flexible, radio-opaque rubber cannula has the proportions of the standard metal cannula (10F, 9 centimeters). It is calibrated in centimeters, with the rubber covered by a special lacquer to facilitate smoother passage through brain tissue. Its lightweight metal hub is fitted with an airtight, watertight stopcock. The tube has a removable stilet.

The cannula may be left in place during x-ray examination and surgery—may be left in the ventricle as a safety valve for 24 to 36 hours following craniotomy—has served as a lifesaving measure for gradual decompression in acute intracranial pressure in comatose patients. Air release after ventriculography requires a mere opening of the stopcock.

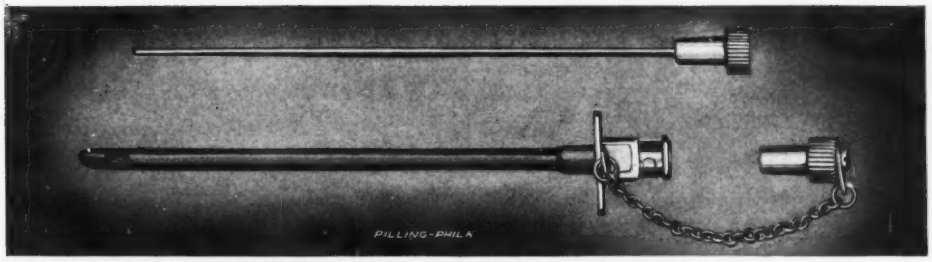
Each, *$14.00*

Instrument Makers To The Profession Since 1895

V. Mueller & Co.

330 SOUTH HONORE STREET, CHICAGO 12, ILLINOIS

BRANCHES NOW IN ROCHESTER, MINN.—DALLAS AND HOUSTON, TEXAS



RUBBER VENTRICULAR CANNULA & INDWELLING CATHETER

Designed by Michael Scott, M.D , Philadelphia

For Ventricular Tapping, and Injection or Removal of Air or Fluid

- No danger of damage to brain by movement of scalp
- Original insertion puts catheter in place—less trauma than metal cannula plus catheter
- Cannula-catheter can be left *in situ* for days without unusual irritation
- Especially valuable during a craniotomy—ventricle tapped, cannula left in place, brain retracted, without danger of catheter damage
- Any system may be Luer-locked to the cannula for continuous injection, pressure estimation or drainage

35-1611 Scott Cannula-Catheter, 8 Fr.	6 cm.
35-1612	8 cm.
35-1613	10 cm.
35-1614 set of three:	one each 6, 8 and 10 cm.

Ref: Scott, M.: J. Neurosurg. 10:438 (July) 1953.

THE AUTHENTIC INSTRUMENT IS AVAILABLE ONLY FROM PILLING . . . *ORDER DIRECT:*

GEORGE P. **Pilling** & SON CO.

3451 WALNUT STREET . . . . . PHILADELPHIA

Automatically controlled SERIAL Angiography

Model 110 Universal Automatic Seriograph

Here is automatically controlled serial angiography with highest quality, standard size pictures and standard developing procedures!

Full control by Radiologist permits test pictures and delays during picture cycle. The 12 individual cassettes, each with its own set of screens, give you 11" x 14" (or smaller) standard pictures for adult angiographic studies.

Seriograph operation is equally efficient in the horizontal or vertical position. Automatic time selector gives ½ to 2 second infinitely variable interval.

For details of these and many other outstanding features of the new Model 110 Universal Automatic Seriograph write to:

The **Automatic Seriograph Corp.**

4910 Calvert Road • College Park • Maryland

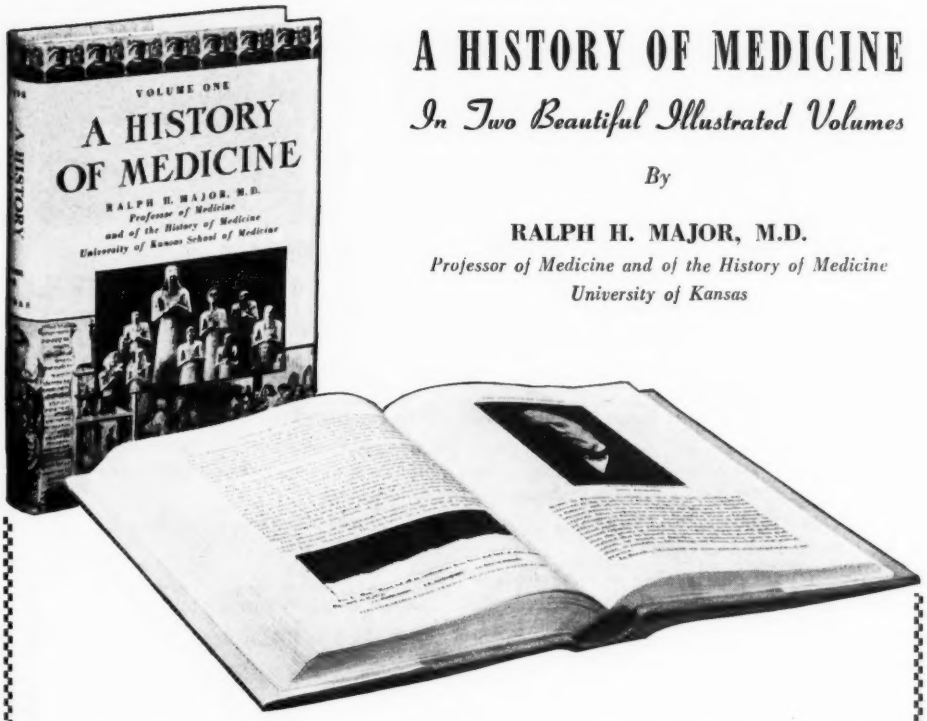
A HISTORY OF MEDICINE

In Two Beautiful Illustrated Volumes

By

RALPH H. MAJOR, M.D.

Professor of Medicine and of the History of Medicine
University of Kansas


VOLUME ONE
A HISTORY OF MEDICINE
RALPH H. MAJOR, M.D.
Professor of Medicine
and of the History of Medicine
University of Kansas School of Medicine

SPANNING *the rich traditions of centuries of medical history, this fresh, new two-volume narrative brings to life the great men and women who developed the healing art from prehistoric times to the middle of the 20th Century.*

Written in terms of the famous individuals who molded medical history, Dr. Major's work is designed to aid the medical student and the medical practitioner in appreciating and learning from the wealth of experience which preceded today's medical miracles.

Profusely illustrated with more than 400 photographs, drawings and reproductions, many taken or collected by the author, "A History of Medicine" traces the mainstream of medical history in a highly readable connected narrative. Each chapter leaves to the serious student further research possibilities through supplementary biographical addenda suggesting detailed readings in the lives of individuals cited in the text.

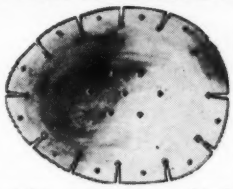
- **Original documents**
- **Photographs taken in historic places**
- **Author has studied in the original language of medicine: *Italian, French, German, English, Latin and Greek***
- **Unpublished material**
- **Represents more than 50,000 miles of personal pilgrimages**
- **Hitherto untranslated works of immense value to an understanding of modern medical science**

1088 pages in 2 volumes 415 illustrations Sent on approval $14.50 postpaid

CHARLES C THOMAS · PUBLISHER · SPRINGFIELD · ILLINOIS

PRE-FORMED SKULL PLATES

...save operating time
...provide finer cosmetic restorations



Nudell's pre-formed plates for replacement of damaged portions of the cranium are highly endorsed by many neurosurgeons. Available in stainless steel or tantalum in 10 different sizes and shapes, including a plate for the right or left supraorbital ridge. Only tons of hydraulic pressure applied by master dies can produce the smooth, even curvatures and intricate shaping of these plates. Slots on the outer edge allow for fast final shaping by the neurosurgeon. Perforations permit growth of granulation tissue.

When large areas are involved or the finest cosmetic appearance is of paramount importance, we recommend your consideration of a custom-made plate. For literature and price list write—

UZA NUDELL
Technician to Neuro-Surgeons
125 WEST 45TH STREET • NEW YORK 36, N.Y.

Simplex
PENTOCRYL
Self Curing Acrylic Resin
for
CRANIOPLASTY

For supplies and full information write to the manufacturers, Dental Fillings Limited, 49 Grayling Road, London N. 16, England.

The Philadelphia General Hospital offers one year combined residencies in Neuropathology and Clinical Neurology for candidates for certification by the American Boards of Neurologic Surgery, Psychiatry and Neurology.

Applicants accepted July 1 and January 1.

For details, apply to: Office of Medical Education, Philadelphia General Hospital, Philadelphia 4, Penna.

New Book

OF SPECIAL INTEREST

Cerebrovascular Disease

By James Peter Murphy, M. D.

Assistant Clinical Professor of Neurological Surgery, George Washington University School of Medicine. 408 pages; 128 illustrations. $12.00

IN THIS new book it has been the author's endeavor to present the clinical tools which will enable the attending physician to deal more effectively with the cerebrovascular accident patient.

Dr. Murphy stresses the important point that *all "strokes" are not alike* and that careful evaluation of the individual patient is the key to favorable prognosis. Every skill and device capable of narrowing the diagnosis is offered in explicit detail, including *valuable differential diagnosis tables.*

Meeting the *therapeutic needs* of the cerebrovascular accident patient is one of the book's major features. All established methods are given—*medical, neurosurgical, and physical.* Surgical procedures are described and illustrated. For instance, stellate block; carotid ligation; subtemporal decompression; and craniotomy.

The illustrations are particularly noteworthy, being artist-perfected copies of originals by the author himself.

---------- ORDER FORM ----------

THE YEAR BOOK PUBLISHERS, INC.
200 East Illinois Street
Chicago 11, Illinois

Please send me the following book for 10 days' examination.

☐ Murphy's Cerebrovascular Disease, $12.00

Name .....Street .....

City .....Zone .....State .....

JNS 1-55

MEASURE

Sensory Changes

The Bio-Thesiometer® enables you to make *improved* sensory tests because it *MEASURES* sensory changes. The results are expressed in numbers as they are in measuring blood pressure, temperature or other quantitative tests.

In addition to giving you quantitative and diagnostic data in your first test, owners emphasize "it is also valuable in following the progress of the disease, whether there is improvement or lack of improvement."

The Bio-Thesiometer® test replaces qualitative and non-standardized tests of sensation. The results are expressed in relative or absolute units which have exactly the same meaning to all those making such tests.

The Bio-Thesiometer is a new tool for the diagnosis of many conditions in which the nervous system or vascular system is involved. May we send you complete information? Write to

BIO-MEDICAL INSTRUMENT COMPANY
24 Munn Road
NEWBURY, OHIO

CONNECTIONS OF THE FRONTAL CORTEX OF THE MONKEY

By

WENDELL J. S. KRIEG

Professor of Anatomy
Northwestern University Medical School

PRESENTING *entirely new data*, this volume is an analysis, reconstructions, and synthesis of the connections worked out in the cortical lesions of seventy monkeys. The subject is the last in the anatomy of the body to be worked out and forms a sizeable block of the subject-matter of neuroanatomy. The neuroanatomy usually studied virtually ends with the brain stem, not because the cerebrum is unimportant, but because it is poorly known.

The NEUROPHYSIOLOGIST needs such information *to explain his results* and *to set experiments.*

Illustrated by
THE AUTHOR

The illustrations on the jacket represent the four phases of research that went into this book.

The author is well qualified to write on this subject. Culminating twenty-five years' preoccupation with the connections of the brain, for the past ten years he has been intensely engaged on an experimental study of the connections of the cerebral cortex.

This work will be a *stimulus* and a *data-source* to scientific workers and to thinkers who must be in the vanguard. Some notions of brain mechanism are no longer tenable, new interpretations are given, and fresh approaches suggested.

320 pages 186 illustrations $10.50, postpaid

CHARLES C THOMAS • PUBLISHER
Springfield • Illinois

ANEURYSMS OF THE MIDDLE CEREBRAL ARTERY

REPORT OF SEVEN OPERATIVE CASES; REVIEW OF LITERATURE; EVALUATION OF SURGICAL THERAPY

DANIEL PETIT-DUTAILLIS, M.D.,* AND HAL W. PITTMAN, M.D.†

Department of Neurological Surgery, Hôpital de la Pitié, Paris, France

(Received for publication August 2, 1954)

INTRACRANIAL arterial aneurysms are not a homogeneous group, and it has recently become apparent that any report dealing with their treatment must consider them according to their location. Since aneurysms of the middle cerebral artery constitute approximately 30 per cent of all intracranial arterial aneurysms[14] and since they present a particular challenge to the neurosurgeon, we thought it appropriate to present our own experience with these aneurysms and to analyze the material published by others.

It cannot be disputed that intracranial aneurysms are serious lesions. A patient entering the hospital with a subarachnoid hemorrhage has roughly a 50 per cent chance of survival without surgical treatment, and of those discharged, about one fourth die of recurrent hemorrhage and one fourth remain disabled.[9,12,15]

CASE MATERIAL

The case material, which is summarized in Table 1, represents all the patients with aneurysm of the middle cerebral artery admitted to the neurosurgical service of the Hôpital de la Pitié since its foundation. With one exception, Case 1 operated on in 1946, all the patients postdated the reorganization of the neurosurgical service in 1948 by the senior author. Nine patients were operated upon; 7 of these had obliteration of the aneurysmal sac, and the other 2 were subjected to evacuation of the hematoma only. The surgical procedures were carried out by the senior author or one of his assistants (Dr. G. Guiot, Dr. J. Pecker, Dr. Y. LeBesnerais, Dr. J.-C. Clément). One patient was discharged without operation, and 2 others died before surgery.

Obliteration of Aneurysm. Seven patients were treated in this fashion. Time intervals between the most recent subarachnoid hemorrhage and operation were 2 months, 36 days, 12 days, 3 months, 1 day, 22 days, and 2 days. Hypotension in the neighborhood of 60 to 80 systolic by the use of vasodepressor drugs was employed in all but the first case. In 5 instances, the base of the aneurysm was occluded by one, sometimes two, clip(s), and in another

* 12, Avenue Lowendal, Paris VII, France.

† Foreign assistant in Neurosurgery, Faculté de Médecine, Paris. Present address: 102 Walnut Street, Fairmont, North Carolina.

TABLE 1

Authors' 12 cases of aneurysm of the middle cerebral artery

Method of Treatment	No. of Cases	Well	Improved	Dead
Occlusion of aneurysm	7	3	2	2
Evacuation of hematoma only	2			2
Operation declined	1	1		
Died before surgery	2			2
Totals	12	4	2	6

by a silk suture. One aneurysm was not pedunculated and required a row of clips across the dome. An intracerebral hematoma of sizable proportions was evacuated in 3 instances and a subdural hematoma in 1.

Transient contralateral hemiplegia appeared during the course of the operation in 2 instances. In Case 6 this was presumably caused by spasm because it regressed after vasorelaxation and in Case 4 by impairment of circulation through the parent artery because it immediately disappeared after a higher placement of the clip on an arteriosclerotic aneurysm.

Case 1. (Aneurysm clipped; well 8 years later.)

H.C., 21-year-old female, was admitted to the Hôpital de la Pitié May 28, 1946 with severe generalized headache for 1 month, and diplopia and ptosis of the right eyelid for 3 weeks.

Examination revealed only a complete right oculomotor nerve paralysis. Spinal fluid was yellow. Arteriogram revealed a small aneurysm near the bifurcation of the right internal carotid artery.

At craniotomy (Dr. G. Guiot) 2 months after hemorrhage the aneurysm was seen to arise from the middle cerebral artery, and a single clip was placed on its base. Arterial bleeding began immediately and was controlled by packing the operative field with gelfoam.

The postoperative course was uneventful, the 3rd nerve lesion regressing completely. The patient was in perfect health 8 years after the operation and had married and given birth to three children.

Case 2. (Aneurysm clipped; postoperative extradural hematoma; death 64 days after operation.)

M.D., 28-year-old female, had sudden severe headache for one hour on Nov. 19, 1952 and similar sudden severe headache 6 days later associated with coma for 6 hours, generalized convulsions, projectile vomiting, meningeal signs, and bloody CSF. Improvement followed, but another attack of headache, coma and convulsions occurred December 12.

Arteriogram at the Pitié revealed an 8-mm. aneurysm of the left middle cerebral artery 2 cm. from its origin.

At operation (Dr. Petit-Dutaillis) 36 days after the second subarachnoid hemorrhage, a liquefied subdural hematoma was evacuated and the aneurysm was clipped with a single silver clip at the base, with the B.P. reduced to 90 systolic by ganglioplegics.

Immediately after the procedure the patient was completely lucid and talking

rationally. Several hours later, a state of decerebrate rigidity developed rapidly, and at reintervention 45 minutes afterward a large extradural hematoma was evacuated. The decerebrate state disappeared the next day, but returned 2 days later after an anoxic episode caused by a suppurative pulmonary complication. It continued to be elicitable by painful stimuli until March 10, after which time the patient was flaccid and totally unresponsive until death in hyperthermia on March 22, 1953, 64 days postoperatively.

Autopsy revealed the aneurysm satisfactorily clipped, the middle cerebral artery distal to the clip patent, and the entire cerebrum and brain stem riddled by petechial hemorrhages.

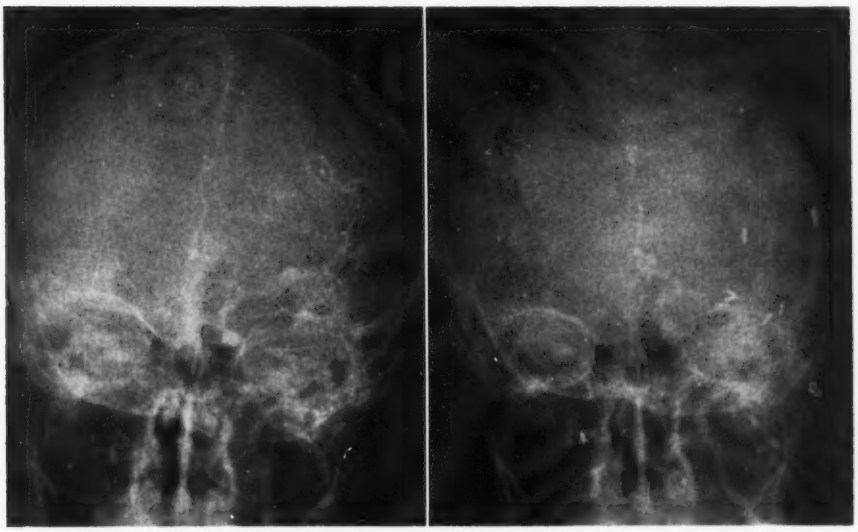


FIG. 1. *Case 3.* Pre- and postoperative arteriograms showing aneurysm of middle cerebral artery satisfactorily clipped.

Case 3. (Aneurysm clipped and large hematoma evacuated; mild aphasia and hemianopsia 1 year later.)

F.D., 46-year-old male, was admitted to the Pitié on June 11, 1953, 1 week after an episode characterized by coma of 2 hours' duration and complete right hemiplegia. Bloody CSF, stiff neck, aphasia, and right hypesthesia were noted; the hemiplegia improved. Arteriography revealed a lobulated 6×13 mm. aneurysm of the left middle cerebral artery 2 cm. from its origin and, in addition, spasm of the intracranial portion of the left internal carotid artery and its branches.

At operation (Dr. Petit-Dutaillis) 12 days after the subarachnoid hemorrhage, the narrow neck of the aneurysm was occluded by a single silver clip with the B.P. reduced to 70/50 by ganglioplegics. Atheromatous plaques were seen on the internal carotid artery and on the aneurysm. A large hematoma was evacuated.

The postoperative course was complicated by a *Staphylococcus* wound infection and bilateral pulmonary tuberculosis, but neurological recovery was progressive so

that 1 year postoperatively, there persisted only mild aphasia and a right homonymous hemianopsia. Arteriogram 2½ months following operation (Fig. 1) shows absence of the aneurysm and patency of the middle cerebral arterial system.

Case 4. (Aneurysm clipped; well 10 months later.)

A.L., 46-year-old male, was admitted to the Pitié on Sept. 15, 1953, with a history of sudden intense headache 2 months previously immediately following a spell of coughing. Stiff neck and bloody spinal fluid were associated, and right temporal headache persisted. Frequent headaches and vertigo had been present for 3 years. Past history included a similar attack of right-sided headache with loss of consciousness in 1944.

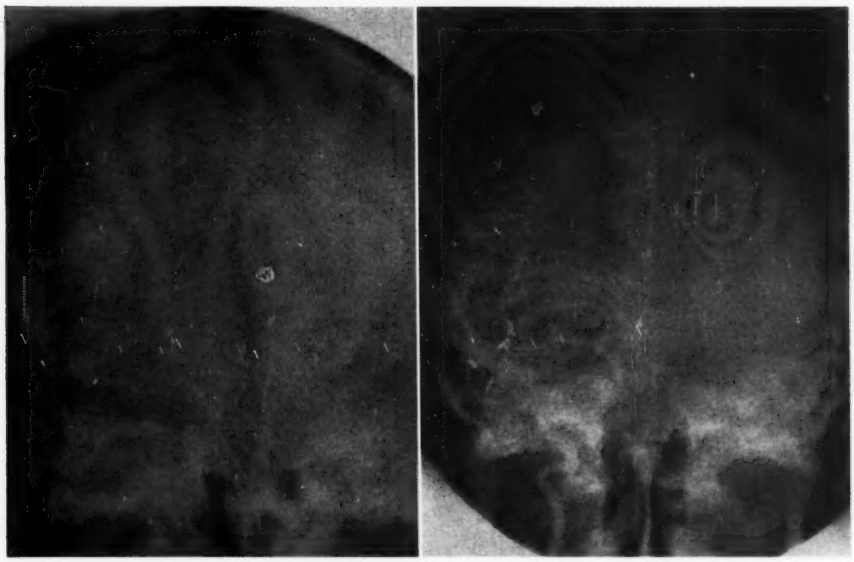


FIG. 2. *Case 4.* Pre- and postoperative arteriograms showing aneurysm of middle cerebral artery satisfactorily clipped.

Examination revealed only left facial weakness. Arteriogram disclosed a bilobate 8×12 mm. aneurysm of the right middle cerebral artery 3½ cm. from its origin.

Operation (Dr. Petit-Dutaillis) was performed 3 months after subarachnoid hemorrhage with B.P. reduced to 100/80 by ganglioplegics. Two clips were required to obliterate the wide base of the aneurysm, which contained a small atheromatous plaque near its dome. The use of local anesthesia permitted the early detection of a contralateral Babinski sign and of a supervening complete hemiplegia about 15 minutes after application of the clips. The lowermost clip, flush with the parent artery, was removed and replaced slightly higher; the hemiplegia began to regress immediately and was completely gone before closure of the scalp.

Postoperative course was smooth, and the patient resumed his occupation as a butcher, working 16 hours a day. He has continued well for 10 months except for

two grand mal seizures and a feeling of not being entirely up to par. Arteriogram 2 months after operation (Fig. 2) shows nonfilling of aneurysmal pouch.

Case 5. (Aneurysm clipped day following second subarachnoid hemorrhage; hematoma and cerebral softening found at operation; minimal aphasia and slight hemiparesis 4½ months later.)

L.P., 29-year-old female, experienced a sudden coma on Oct. 21, 1953, preceded by moderately severe occipital and nuchal headache for several days and by milder headaches for several years. At another hospital, lumbar puncture yielded bloody CSF, and neurological examination showed nuchal rigidity and coma which improved to a state of confusion with pronounced aphasia.

On admission to the Pitié, on Nov. 2, 1953, only motor aphasia and indifference were found on examination. On the evening of November 4, the patient had two episodes of decerebrate rigidity, each lasting 10 minutes and followed by profound coma and right hemiplegia, both of which symptoms improved rapidly. Arteriography the next day showed a 6×8 mm. aneurysm on the left middle cerebral artery 1½ cm. from its origin. Other arteriographic findings were spasm of the left internal carotid artery and a hematoma.

At operation (Dr. Petit-Dutaillis) immediately afterward, the aneurysm was actively bleeding from several points and required 4 or 5 clips across its dome because its neck was too broad for clipping. Intracerebral hematoma and frontal lobe softening were also found. The aneurysm was approached with the systolic pressure at 60 by means of ganglioplegics.

The patient made rapid progress postoperatively, and 4½ months later she showed only minimal motor aphasia and slight weakness on the right.

Case 6. (Aneurysm ligated; well 1 month later.)

J.M., 48-year-old male, previously healthy, suddenly fell to the floor unconscious for 1–2 minutes; severe headache appeared but diminished in intensity during the 3 weeks prior to admission to the Pitié.

Examination revealed mild meningeal signs and slight left facial weakness. Arteriogram disclosed a 7 mm. aneurysm on the right middle cerebral artery 2½ cm. from its origin.

On May 14, 1954, 22 days after subarachnoid hemorrhage, under local anesthesia and with the B.P. lowered slightly with ganglioplegics, the base of the aneurysm was ligated with a silk suture (Dr. Petit-Dutaillis). About 10–15 minutes later, the sensorium became obtunded and a left hemiplegia developed. The theory that these symptoms were caused by arterial spasm seemed borne out by their complete disappearance in 12 hours after treatment with papaverine intramuscularly and continuous slow drip of procaine intravenously.

The postoperative course was uncomplicated, and the patient was discharged in perfect health 3 weeks after operation.

Case 7. (Arterial hypertension; aneurysm clipped 2 days following hemorrhage; enormous intracerebral hematoma; death 4 days postoperatively.)

R.G., 61-year-old hypertensive (200 to 240 for 8 years) female, presented mild clouding of the sensorium and mild meningeal signs immediately after a fall from a bicycle. Twenty-four hours later, left hemiplegia and left hemianopsia had developed, and the following day, May 29, 1954, the patient was admitted to the Pitié.

Examination revealed a B.P. of 160/90 and hypertensive fundi in addition to the signs mentioned. Arteriography (Fig. 3) showed a 5 mm. aneurysm on a major branch of the right middle cerebral artery and spasm of the 3 cm. segment proximal to the aneurysm.

Operation was performed as an emergency procedure (Dr. LeBesnerais) on the day of admission under local anesthesia with the B.P. lowered to about 100/50 with ganglioplegics. The base of the aneurysm was clipped and the pouch was coagulated. An enormous hematoma extending to the occipital lobe was evacuated.

Postoperatively the patient responded slightly but remained obtunded and hemiplegic. She gradually lost consciousness and died on the 4th postoperative day. Autopsy revealed the aneurysm properly clipped, and no cause of death was discernible.

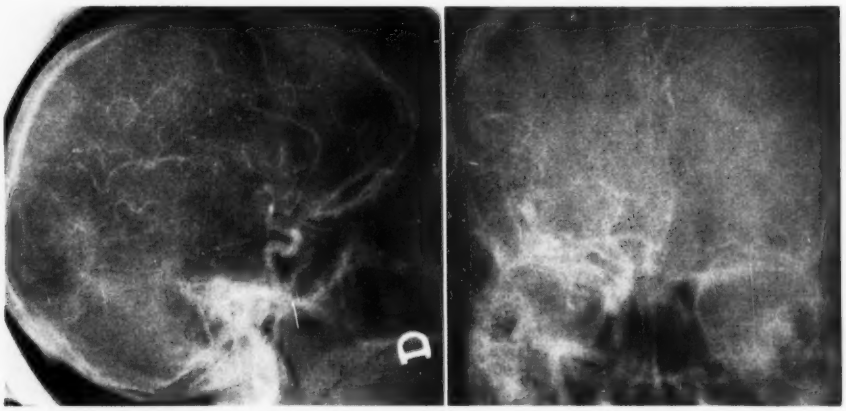


FIG. 3. *Case 7.* Arteriogram showing middle cerebral artery aneurysm, segmental spasm of middle cerebral artery, and temporal hematoma.

Of 7 patients operated upon, 3 are well, 2 are improved in comparison with their preoperative state, and 2 died postoperatively. One postoperative death was the result of an extradural hematoma; the other occurred in a hypertensive female with an enormous intracerebral hematoma. Autopsy in both instances showed the aneurysm properly clipped and the middle cerebral artery patent distal to the aneurysm. The 2 patients who are improved but not in perfect health are disturbed principally by mild aphasia caused by intracerebral hematoma. Postoperative arteriograms in Cases 3 and 4 showed absence of the aneurysm and patency of the entire middle cerebral arterial system. Follow-up periods are 8 years, 1 year, 10 months, 4½ months, and 1 month.

Evacuation of Hematoma. Two patients, aged 45 and 64, one severely hypertensive, had temporal trephination and evacuation of an intracerebral hematoma 2 and 13 days after hemorrhage. One patient had more complete evacuation of the hematoma through a bone flap 5 days after trephination.

Both patients were hemiparetic, aphasic, and mentally obtunded, and both had been demonstrated to have intracerebral hematomas by arteriography. Both patients died soon after operation, one on the operative day, the other on the 6th postoperative day. Autopsy in both instances revealed aneurysm of the middle cerebral artery in the sylvian fissure and recurrence of a large intracerebral hematoma.

CASES IN LITERATURE OF SURGICALLY TREATED MIDDLE CEREBRAL ARTERY ANEURYSMS

In the literature we have been able to find 76 cases of aneurysm of the middle cerebral artery subjected to surgery. An additional case reported by de Sèze, Feld, and Guiot[19] is included in this report as Case 1.

Table 2 shows that of the 76 patients, 50 (66 per cent) were successfully treated, 19 (25 per cent) died, and 7 (9 per cent) survived with disability. The surgical procedures employed were occlusion of the neck of the aneurysm in 29 cases, wrapping of the aneurysm with muscle in 12 cases, occlusion of the parent artery in 6 cases, and ligation of the carotid artery in the neck in 18 cases. Nine patients had only evacuation of the intracerebral hematoma, and 2 were explored but not treated. Practically all patients had had recent subarachnoid hemorrhage, but the interval of time between subarachnoid hemorrhage and operation was not stated in a sufficient number of cases to permit statistical evaluation.

Occlusion of Neck of Aneurysm. The largest number of patients were treated by occlusion of the neck of the aneurysm either by silver clips or nonabsorbable ligature. Most authors preferred silver clips because the possibility of tearing the neck of the sac is apparently less. Two aneurysms tore on application of clip and were treated by packing with muscle. It has been remarked that placing the clip exactly flush with the parent artery is not necessary. In a few cases, the aneurysm was excised, but this is generally not recommended because it loosens the clip. In 1 case the aneurysm was simply coagulated, and in 2 others, coagulation of the aneurysm was used to supplement clipping of the neck.[21] Matas endaneurysmorrhaphy was employed in 1 case when the aneurysm ruptured during the operation.[5]

Of 29 patients who were treated by ligation of the neck of the aneurysm, 24 (83 per cent) are well or improved, 3 (10 per cent) are dead, and 2 (7 per cent) are disabled.

Ligation of Parent Artery. Of 6 patients in whom the feeding artery was ligated and the aneurysm thus excluded from the cerebral circulation, 4 survived, 1 died, and 1 remained hemiparetic. This technique should be limited to lesions located elsewhere than the main trunk of the artery and, in so far as possible, to branches that are relatively unimportant as nourishing arteries of the motor area or language center. Aneurysms of mycotic etiology usually occur distally and therefore are amenable to treatment by ligation of the feeding artery.

Wrapping of the Aneurysm. In 12 cases the aneurysm was wrapped with

TABLE 2

Cases of aneurysm of the middle cerebral artery reported in the literature, showing method of treatment and results

Author	No. of Operative Cases	Neck of Aneurysm Occluded					Aneurysm Wrapped With Muscle				Parent Artery Occluded				Cervical Carotid Ligation Only				Evacuation of Hematoma Only			Intracranial Exploration Only	
		No. of Patients	Well or Improved	Hemiparetic	Aphasic	Dead	No. of Patients	Well or Improved	Hemiplegic	Dead	No. of Patients	Well	Hemiparetic	Dead	No. of Patients	Well or Improved	Hemiplegic	Dead	No. of Patients	Well	Dead	No. of Patients	Dead
Norlén & Olivecrona[16]	17	13	12			1	4	4							4	1	3		2		2		
Falconer[8]	10	1	1				3	2	1														
Laine, Soots & Delandsher[13]	7	1	1				3	2		1	1	1			1			1				1	1
Steelman, Hayes & Rizzoli[20]	7	4	4				1	1							2	2							
Bassett, List & Lemmen[3]	6										3	2	1		2	2			1	1			
Campbell & Burkland[5]	6	5	2	1	1	1					1	1											
Wechsler, Gross & Cohen[24]	6	1				1									5	5							
Dandy[6]	5										1			1	1			1	2		2	1	1
Hamby[10]	3																		3		3		
Swain[21]	3	3	3																				
Bassett[2]	1	1	1												1			1					
Black & German[4]	1																						
Dott[7]	1						1	1															
Hermann, Obrador & Dott[11]	1														1			1	1		1		
Hyland[12]	1																						
Thévenard & Guiot[23]	1														1	1							
Totals	76	29	24	1	1	3	12	10	1	1	6	4	1	1	18	11	3	4	9	1	8	2	2

Summary of all cases:

Well or improved	50
Hemiplegic, hemiparetic, or aphasic	7
Dead	19

hammered muscle. Of these patients, 1 died, 1 was rendered hemiplegic, and 10 successfully withstood the procedure and remained well during the generally short observation periods. The permanence of the cure remains to be determined by longer follow-up, but it appears probable that enveloping the aneurysm in muscle affords additional support for the weakened wall.

Evacuation of Hematoma Only. Of the 9 cases in which the hematoma was evacuated but the aneurysm not treated, death occurred in 8 and 1 patient remained well 9 years later. At least 3 patients died of recurrent hemorrhage from the aneurysm after evacuation of the clot, presumably because of removal of support from around the weakened wall of the sac. *It therefore seems inadvisable to evacuate a hematoma without attempting a direct attack on the aneurysm.* In many instances, surgical exposure of the aneurysm is greatly facilitated by removal of an intracerebral hematoma, and evacuation of such clots should never be omitted during the course of a definitive attack on the lesion.

Intracranial Exploration Only. Of 2 patients explored intracranially but not treated, both died. The aneurysm was not found in one case and was associated with cerebral softening in the other.

Cervical Carotid Ligation. There were 18 patients treated by carotid ligation in the neck. One of these[2] had ligation of the carotid artery intracranially also, as treatment for an associated internal carotid aneurysm. Six patients, not included among these 18, had ligation of the carotid artery in the neck in addition to intracranial attack on the aneurysm. The ligation in most cases was of the common carotid and in practically all cases was complete. In 3 cases, the artery was divided after ligation. Efficiency of collateral cerebral circulation was determined in all cases before ligation, and complications were generally those that appeared after an interval of a day or several days, and are therefore presumed to have been caused by thrombosis or embolism rather than by the immediate decrease in cerebral circulation. Four of these 18 patients died, and 3 were rendered hemiparetic; thus a total of 7, or 39 per cent, suffered death or serious complications. The remaining 11 are well or improved. The shorter the interval between subarachnoid hemorrhage and carotid ligation, the greater was the incidence of complications.

DISCUSSION

Intracranial Operation. The use of vasodepressor drugs to lower arterial pressure for intracranial procedures, as one of us has shown since 1951 with Guiot and Damoiseau,[17,18] is a very important adjunct to the surgical management of arterial aneurysms because it permits exposure of the sac with less danger of rupture. Severe hemorrhage consequent to rupture may be safeguarded against by occlusion intracranially of the middle cerebral artery proximal to the aneurysm by use of removable clips introduced by Olivecrona or by a noose of silk as recommended by Campbell and Burklund.[5] Caution must be used with these techniques to avoid occlusion of the main trunk of the middle cerebral artery longer than several minutes, or ischemic brain

damage will result. If craniotomy is undertaken in a case in which arteriography has shown that clipping of the base would be doubtful, the carotid artery in the neck may be ligated beforehand, or it may be encircled by a removable noose which can be quickly tightened in case of need. Prevention or alleviation of arterial spasm is important and can best be managed by applying papaverine directly to the arterial wall at operation or later by intracarotid injection of papaverine or by blocking the stellate ganglion.

Selection of the proper time for operation is very important and remains controversial. It is best to wait until the patient's condition, if not improved, has at least been stabilized, for operative complications are greater in the period immediately following rupture. Unnecessary delay is to be avoided because the incidence of recurrent rupture is particularly high in the first 2 weeks. Recurrence of the hemorrhage or the coexistence of an intracerebral hematoma constitute, according to us, indications for immediate operation.

Cervical Carotid Ligation. Most surgeons feel that the common carotid is the safer artery to ligate, whereas others ligate the internal carotid initially. Some ligate first the common carotid artery and later the internal. Sweet, Sarnoff, and Bakay[22] have shown that the internal carotid artery does not always receive collateral circulation from the external after occlusion of the common carotid.

Carotid ligation as a method of treatment cannot be properly evaluated from existing data. Long-term follow-ups are essential. Two very important questions remain to be answered, namely: (1) does cervical ligation give assurance of a permanent cure, and (2) will deprivation of one of the carotid arteries feeding the circle of Willis result in important deficit in nutrition of cerebral tissue as age advances. Answer to the second question can be arrived at by prolonged follow-up with repeated neurological and psychological evaluation. As to the first question, it seems highly probable that cervical ligation affords some protection against subsequent rupture of the aneurysm. The pressure within the middle cerebral artery is known to be reduced by carotid ligation in the neck. Sweet, Sarnoff, and Bakay[22] have shown that in the proximal segment of the internal carotid artery, the systolic pressure drops to 51 per cent shortly after its occlusion. Subsequently, Bakay and Sweet[1] showed that pressures in small intracranial arterial branches do not differ markedly from those in the internal carotid artery and that pressures following carotid ligation are decreased the same percentage in all intracranial arteries down to those 0.4 mm. in diameter. They suggest from these data that carotid ligation should be an equally effective therapeutic measure, regardless of whether the intracranial aneurysm is located proximally on large trunks or distally on ramifications, and that measurements of the pressure in the carotid artery in the neck distal to occlusion may be used to prognosticate the degree of effectiveness. Pressure drops to about half are desirable, and those in the range of 30 per cent of the original level indicate that the collateral circulation is poor and probably will not permit complete ligation of the carotid artery in a single stage.

That aneurysms can shrink in size after the pressure within them is reduced is indicated by marked improvement of oculomotor nerve symptoms following carotid ligation for aneurysms of the internal carotid artery. Available statistics suggest that aneurysms so reduced in size undergo some sort of healing process and become more resistant to rupture. Testimony that carotid ligation is not always a cure for middle cerebral aneurysms comes from a report by Black and German[4] of a 34-year-old patient whose right common carotid artery was ligated for an aneurysm of the supraclinoid portion of the internal carotid artery. Twelve days later, a fatal hemorrhage occurred from an aneurysm of the right middle cerebral artery which was to have been attacked later by direct approach.

Comparison of Intracranial Operation and Cervical Carotid Ligation. The personal experience of the authors and the results obtained from a candid review of the literature lead the authors to the conclusion that intracranial clipping of the aneurysm is the procedure of choice in the treatment of intracranial aneurysms situated on the middle cerebral artery. Whereas aneurysms of the cavernous portion of the internal carotid artery, for example, are inaccessible to direct approach and require the neurosurgeon to be content with carotid ligation, aneurysms of the middle cerebral artery are located particularly conveniently for the neurosurgeon. Because of the ease of surgical exposure of the middle cerebral artery and in view of the low operative mortality, the authors feel that failure to make a direct surgical attack on the aneurysm is not justified. The authors are not content to reduce the pressure within the aneurysm to half its original value by cervical carotid ligation when a definitive cure can be accomplished by obliteration of the neck of the aneurysm. Any neurological disability persisting after clipping of the aneurysm is rarely greater than pre-existing neurological deficit and is usually the result of damage caused by an intracerebral hematoma.

Strengthening the argument in favor of ligating the aneurysm intracranially as opposed to tying the carotid in the neck are the comparative mortalities for the two procedures. Of the 29 patients from the literature treated by obliteration of the aneurysm plus our 7 cases, 5 died, giving a mortality rate of 14 per cent, and 2 were disabled. By comparison, cervical carotid ligation gave a mortality rate of 4 out of 18, or 22 per cent, with an additional 3 patients disabled. If we group the three categories in which a definitive attack was made on the aneurysm intracranially, namely: obliteration of the neck of the aneurysm, wrapping of the aneurysm, and ligation of the parent artery, we arrive at a collective mortality of 13 per cent and a disability of 7 per cent. There has been no report of a recurrence of rupture of an aneurysm treated by one of these three methods; whereas, at least one middle cerebral aneurysm has ruptured subsequent to cervical carotid ligation. Clipping of the aneurysm and ligation of the parent artery are curative procedures; if, however, at intracranial operation, the aneurysm has not permitted either of these procedures and the surgeon has had to be content with simple wrapping of the aneurysm, it is wise also to ligate the carotid artery in the neck.

SUMMARY

The authors present 7 cases of aneurysm of the middle cerebral artery obliterated by silver clips or silk suture with 5 successful results. Two patients with middle cerebral artery aneurysms subjected only to evacuation of an intracerebral hematoma died.

Seventy-six cases of aneurysm of the middle cerebral artery collected from the literature are analyzed. Obliteration of the aneurysm was the procedure that gave the highest percentage of successful results, specifically, 83 per cent. Ligation of the carotid artery in the neck gave a mortality of 22 per cent. The authors conclude that the preferred method of treatment is intracranial attack with obliteration of the aneurysm whenever possible.

REFERENCES

1. Bakay, L., and Sweet, W. H. Cervical and intracranial intra-arterial pressures with and without vascular occlusion. *Surg. Gynec. Obstet.*, 1952, *95:* 67–75.
2. Bassett, R. C. Multiple cerebral aneurysm. Report of a case. *J. Neurosurg.*, 1951, *8:* 132–133.
3. Bassett, R. C., List, C. F., and Lemmen, L. J. Surgical treatment of intracranial aneurysm. *Surg. Gynec. Obstet.*, 1952, *95:* 701–708.
4. Black, S. P. W., and German, W. J. The treatment of internal carotid artery aneurysms by proximal arterial ligation. A follow-up study. *J. Neurosurg.*, 1953, *10:* 590–601.
5. Campbell, E., and Burklund, C. W. Aneurysms of the middle cerebral artery. *Ann. Surg.*, 1953, *137:* 18–28.
6. Dandy, W. E. Intracranial arterial aneurysms. *Ithaca, N. Y.: Comstock Publishing Co., Inc.*, 1944, viii, 147 pp.
7. Dott, N. M. Intracranial aneurysms: cerebral arterio-radiography: surgical treatment. *Trans. med.-chir. Soc. Edinb.*, 1933, n.s. *40:* 219–234.
8. Falconer, M. A. The surgical treatment of bleeding intracranial aneurysms. *J. Neurol. Neurosurg. Psychiat.*, 1951, n.s. *14:* 153–186.
9. Hamby, W. B. Spontaneous subarachnoid hemorrhage of aneurysmal origin. Factors influencing prognosis. *J. Amer. med. Ass.*, 1948, *136:* 522–527.
10. Hamby, W. B. Intracranial aneurysms. *Springfield, Ill.: Charles C Thomas*, 1952, xxi, 564 pp.
11. Hermann, K., Obrador, S., and Dott, N. M. Intracranial aneurysms and allied clinical syndromes. Cerebral arteriography in their management. *Lisboa méd.*, 1937, *14:* 782–814.
12. Hyland, H. H. Prognosis in spontaneous subarachnoid hemorrhage. *Arch. Neurol. Psychiat., Chicago*, 1950, *63:* 61–78.
13. Laine, Soots, and Delandsher. Chirurgie des anévrysmes intra-crâniens, à propos d'une série de dix-huit observations. *Lille chir.*, 1951, *6:* 101–110.
14. McDonald, C. A., and Korb, M. Intracranial aneurysms. *Arch. Neurol. Psychiat., Chicago*, 1939, *42:* 298–328.
15. Magee, C. G. Spontaneous subarachnoid haemorrhage. A review of 150 cases. *Lancet*, 1943, *2:* 497–500.
16. Norlén, G., and Olivecrona, H. The treatment of aneurysms of the circle of Willis. *J. Neurosurg.*, 1953, *10:* 404–415.
17. Petit-Dutaillis, D., and Guiot, G. Indications et résultats de l'utilisation de certains ganglioplégiques (hypotension controlée) en chirurgie cérébrale. *J. int. Chir.*, 1952, *12:* 241–252.
18. Petit-Dutaillis, D., Guiot, G., and Damoiseau, B. De l'utilisation de certains ganglioplégiques (hexaméthonium, 92.95 C) et des résultats qu'on en peut obtenir en neuro-chirurgie. *Rev. Path. Comp.*, 1952, *52:* 651–668.
19. de Sèze, S., Feld, M., and Guiot, G. Traitement d'un anévrysme congénital de la sylvienne droite par la fermeture du collet. *Rev. neurol.*, 1947, *79:* 191–193.
20. Steelman, H. F., Hayes, G. J., and Rizzoli, H. V. Surgical treatment of saccular intracranial aneurysms. A report of 56 consecutively treated patients. *J. Neurosurg.*, 1953, *10:* 564–576.
21. Swain, R. D. The surgical treatment of certain intracranial arterial aneurysms. *Surg. Clin. N. Amer.*, April 1948, *28:* 396–404.
22. Sweet, W. H., Sarnoff, S. J., and Bakay, L. A clinical method for recording internal carotid pressure. *Surg., Gynec. Obstet.*, 1950, *90:* 327–334.
23. Thévenard, A., and Guiot, G. Volumineux anévrysme sylvien. *Rev. neurol.*, 1950, *82:* 214–217.
24. Wechsler, I. S., Gross, S. W., and Cohen, I. Arteriography and carotid artery ligation in intracranial aneurysm and vascular malformation. *J. Neurol. Neurosurg. Psychiat.*, 1951, n.s. *14:* 25–34.

DECEREBRATE STATE IN CHILDREN AND ADOLESCENTS*

ROBERT C. L. ROBERTSON, M.D.†, AND CLAUDE POLLARD, JR., M.D.
Division of Neurosurgery, Department of Surgery, Baylor University Medical College, Houston, Texas

(Received for publication August 17, 1954)

CLINICAL signs of decerebrate rigidity in the human are seen frequently, may result from a variety of intracranial lesions in both adults and children, and always have been associated with a more or less hopeless prognosis. The characteristic neurologic signs—profound unconsciousness, extensor rigidity in the arms and legs, bilateral Babinski sign and pin point pupils—indicate a severe lesion at the midbrain level, whereas the presence of flexion of the arms rather than extension and the tonic neck and labyrinthine reflexes of Magnus and de Kleyn have been said to represent a state of decorticate rigidity.[1] Actually, little distinction between decerebrate and decorticate is made clinically and in most instances of brain stem and/or midbrain lesions the rigidity is designated decerebrate, although this is not entirely accurate. Likewise, little distinction is made between "high" and "low" decerebration and perhaps it is not possible to obtain a precise classification in the human since it is rare to encounter an isolated lesion comparable to those produced in experimental animals. The reflexes of Magnus and de Kleyn are interpreted as a sign of the decorticate rather than the decerebrate state because in severe degrees of the decerebrate state the extensor rigidity of the arms may be so marked that the characteristic extension of the "jaw limbs" and flexion of the "skull limbs" are occluded. The constant presence of pin point pupils infers that the maximum lesion is in the region of the mesencephalon.

In clinical practice, most patients are probably in the decorticate state and nearly all of them have one or more associated lesion(s), such as cerebral laceration, intracranial hematoma, increased intracranial tension, vascular hemorrhage or thrombosis, or a degenerative process secondary to anoxia. One rarely sees a single lesion involving the midbrain or brain stem although the clinical syndrome of midbrain interruption of severe degree is not uncommon. Signs of associated injury to the hypothalamus may occur with such phenomena as sham rage, diabetes insipidus, loss of thermal control, reflex chewing, laughing and crying, and marked personality changes, as well as alterations in sleep, blood pressure, pulse, respiration and sphincter control. In addition periodic episodes of profuse sweating and excess production of bronchial secretions are signs of dysfunction of the autonomic nervous system. These are observed in nearly all instances of the decerebrate

* Presented at the meeting of The Southern Neurosurgical Society, Houston, Texas, March 13, 1953.
† Hermann Professional Bldg., Houston, Texas.

TABLE 1

Adults

Case	Age	Etiology	Severity	Duration	Tracheostomy	Result				
						Normal	Improving	Sl. Improv.	Unchanged	Died
1. R.L.F.	21	Motorcycle accident July 29, 1952	3	14 days	+		8 mos.			
2. C.McW.	27	Thrown out of car Jan. 4, 1952	4	9 mos.	−			Minimal 14 mos.		
3. J.B.	29	Hemorrhage cerebellar cyst & vent. Apr. 28, 1952	4	20 hrs.	−					20 hrs.
4. E.V.P.	29	Blow on head from falling pipe Jan. 7, 1952	3	38 days	−			Minimal 14 mos.		
5. J.A.G.	32	Thrown off horse Nov. 23, 1951	4	3 days	+					3 days
6. T.D.	40	Hemorrhage r. int. cap. & vent. May 21, 1952	2	12 hrs.	−					1 day
7. R.R.B.	40	Blow on head by ice bucket Oct. 16, 1951	4	7 mos.	+			Minimal 17 mos.		
8. W.W.W.	42	Automobile accident (bilat. sub. hyd.) Oct. 31, 1951	2	78 days	+					3 mos.
9. W.F.	45	Cardiac arrest—lap. (18 mins.) 1948	4	4 mos.	−					6 mos.
10. C.G.	50	Cardiac arrest—lap. (12 mins.) Jan. 18, 1953	3	1 mo.	+			2 mos.		
11. G.M.A.	51	Thrombosis basilar a. & pon. hem. Mar. 20, 1950	3	3 days	−					3 days
12. A.A.U.	53	Thrown off horse Dec. 7, 1950	2	1 mo.	−					1 mo.
13. A.J.K.	57	Automobile accident Jan. 7, 1951	2	1 mo.	−		26 mos. (Not working)			
						0	2 (16%)	4 (31%)	0	7 (53%)

or decorticate state. Frequently these unconscious patients require tracheostomy.

The present report is a study of the respective fates of an unselected and consecutive group of 13 adults and 13 children. All of them have shown

TABLE 2

Children and adolescents

Case	Age	Etiology	Severity	Duration	Tracheostomy	Result				
						Normal	Improving	Sl. Improv.	Unchanged	Died
1. O.H.	16 mos.	Extradural hematoma from fall Aug. 14, 1952	4	36 hrs.	−					36 hrs.
2. L.S.	2	Hydrocephalus Dec. 9, 1952	4	4 days	−					4 days
3. D.P.	3	Ether convulsion (cleft palate) Nov. 14, 1953	4	2 days	+					2 days
4. J.D.	4	Kicked by mule Sept. 21, 1948	3	24–36 hrs.	−	4½ yrs.				
5. T.E.	7	Struck by car Oct. 22, 1951	3	5 days	−	1½ yrs.				
6. J.G.	7	L. vent. hem. & Jack. conv. Dec. 30, 1951	4	11 days	−	1¼ yrs.				
7. M.H.	7	Fall from upper bunk of bed Oct. 22, 1951	2	10–12 hrs.	−	1½ yrs.				
8. G.G.	7	Struck by car Mar. 25, 1951	4 Sham rage	13 days	+	2 yrs.				
9. J.J.	9	Air rifle wound Sept. 4, 1952	3 Sham rage	12 days	−	6½ mos.				
10. J.R.D.	13	Thrown off motor scooter May 31, 1952	2	6–8 hrs.	−					21 hrs.
11. B.M.	15	Thrown off horse Mar. 15, 1952	4	30 days	−		Marked 1 yr.			
12. M.J.	17	Cardiac arrest—lap. (13 mins.) Nov. 18, 1952	4	46 days	+				4 mos.	
13. J.L.C.	19	Automobile accident July 23, 1952	4	31 days	+			Minimal 8 mos.		
						6 (45%)	1 (8%)	1 (8%)	1 (8%)	4 (31%)

definite clinical signs of more or less complete rigidity, probably most often of high or decorticate type. The two groups of cases are small and are probably of little statistical significance. We have been surprised to find, however, that 6 of the 13 children do not have the serious permanent neurologic impairment usually considered inevitable when the clinical decerebrate state

is definite. In fact, these 6 children recovered to an approximately normal neurological state, a result we have not yet observed in an adult.

Dr. F. K. Bradford allowed us to review about 13 cases of adults in his series, and in most respects they are similar to ours.

As may be noted in Tables 1 and 2, none of the adults has recovered to a degree even approximating his former state, whereas 6 of the 13 children are now normal except for convulsive seizures in the boy kicked by a mule (Case 4) and marked spiking foci in the electroencephalogram of a girl (Case 5) struck by an automobile. The boy shot with an air rifle (Case 9) has recovered from his decerebrate signs but still shows a 6th nerve paresis and personality changes which are improving.

In children the duration of the decerebrate state apparently has not affected the degree of recovery. The 3 children with decerebrate signs for 11, 12 and 13 days recovered as completely as the 3 with signs for 10 hours, 36 hours and 5 days. The 3 with signs for 11, 12 and 13 days required about 3 months to recover from their spasticity, hyperreflexia and bilateral Babinski sign.

In this small series, it is evident that the immediate mortality rate in the children is about the same as in the adults and we believe that the decerebrate state, especially in the young child, is a very dangerous situation. However, if the child survives the immediate neural insult, the outlook for recovery to normal or with minimal neurologic loss is distinctly better than in the adult. The intensity of neurologic signs has not necessarily paralleled the mortality rate since several patients with comparatively mild signs have died, while 3 children with marked signs have made relatively complete recovery.

Likewise in cases in which tracheostomy was done, some of the patients subsequently died, and others who did not have tracheostomy survived. In fact, none of the 6 children who recovered to a normal state required tracheostomy. Nevertheless, we strongly advocate its use in any patient whose respiratory system cannot be easily kept free of secretions by postural drainage and suction. We have often observed the marked increase of rigidity that occurs when the patient with decerebrate signs needs his airway cleared of secretions. Especially is this true in those who have some degree of hypoxia from partial respiratory obstruction. We believe that unnecessary delay in doing a tracheostomy, when it is needed, may very well add to the permanent neurologic deficit if repeated episodes of hypoxia and cyanosis are allowed to occur.

As far as can be determined, all of the children who survived have an intellectual level normal for their age. They are in their usual grade in school. In fact, the boy struck by a car (Case 8) who showed sham rage has proved to be an excellent and outstanding student. The boy (Case 6) who suffered an intraventricular hemorrhage and showed the most marked signs of all the children, now plays the piano as well as originally. He is making normal progress in his music lessons. Our most favorable results have been in pa-

tients about 4 to 15 years of age, but in the late teens the outcome may closely parallel that of the adult patient. So far we have no children under our care who are neurologic wrecks as a result of their once having been in a decerebrate state. Adult patients in such condition are all too common. Not a single one of our adult patients has been able to work; in fact, there are only 2 who can feed and dress themselves and attend to their toilet.

CONCLUSIONS

1. In children who survive the immediate insult, the decerebrate state has a much better prognosis than a comparable lesion in the adult.

2. Six of 13 children and adolescents are relatively normal neurologically compared to none of 13 adults who showed similar decerebrate states.

3. None of the children is a neurologic wreck whereas postdecerebrate vegetation in adults is all too common.

4. In a larger group of cases, the mortality rate from the immediate insult in the child, especially the very young, would probably be as high or higher than in the adult.

5. Tracheostomy is advocated as an early measure to control partial respiratory obstruction caused by secretions and to avoid repeated episodes of hypoxia and cyanosis which may increase permanent neurologic deficits.

REFERENCE

1. Fulton, J. F. Physiology of the nervous system. *New York: Oxford Univ. Press.*, 1949, 3rd ed., x, 667 pp. (see pp. 157–265).

SOME EFFERENT CONNEXIONS OF THE HUMAN FRONTAL LOBE

ALFONSO MARTÍNEZ, M.D.

Laboratory of Neuropathology, Institute of Neurosurgery and Brain Research, Santiago, Chile*

(Received for publication August 24, 1954)

THE principal source of anatomic information on the frontothalamic and thalamofrontal connexions have been the experimental studies made on laboratory animals. The information obtained has been useful especially with preparations stained by the Marchi method to determine the course of the myelinated fibers.

A detailed communication on these connexions in human material, with a study of 7 cases of leucotomy and 1 of lobotomy, was made by Meyer, Beck and McLardy[8] in 1947.

During the last several years various investigators have been using different techniques of silver impregnation for the study of degeneration of fibers and terminations of the central nervous system.[4,5,7] Meyer,[9] in 1949, for the first time analysed human leucotomized brains with the silver impregnation technique of Glees, in order to demonstrate the course of the corticofugal fibers up to the terminations of the cortical nuclei. Beck,[2] in 1950, studied a selected material of 5 cases of leucotomy with Glees' technique and demonstrated that the prefrontopontine bundle originates in the dorsal part and lateral convexity of the prefrontal region. Beck, Meyer and Le Beau,[3] in 1951, in 2 cases of cortical resections in human material, studied the efferent connexions of agranular frontal cortex and described a direct frontohypothalamic pathway terminating in the ventromedial and lateral hypothalamic nuclei; the authors employed the silver impregnation technique of Glees.

Recently, Vaz Ferreira,[10] in 1952, used the sixth formula of Cajal in experimental studies of degeneration of thalamocortical fibers in rats.

According to Meyer, Beck and McLardy,[8] the location of the operation in lobotomy or leucotomy does not have any importance in examining the brain. They state: "An operation on human beings producing a brain lesion unpredictable in precise location and extent is from a purely anatomical point of view an unqualified boon, since each case, and often each hemisphere, presents as it were a separate experiment."

The present paper is based on histological studies of the corticofrontal pathway in 2 cases only: one of bilateral topectomy of area 32 and part of areas 8, 10 and 24 of Brodmann, and the other, a unilateral circumscribed resection of the cerebral cortex and white substance of the frontal lobe in areas 46 and 10.

* Director: Professor A. Asenjo.

MATERIAL AND METHODS

The autopsies were made personally. In Case 1 the brain was removed 5 hours after death. The body was maintained at 0°C. before the autopsy was performed, so that autolitic changes were regarded as unimportant. In Case 2, the brain was removed 1 hour after death.

The brains were fixed without delay by suspension in 15 per cent formalin for 8 hours. Then sections were made and photographed. Blocks which included the thalamus, lentiform, caudate, putamen, and, in Case 2, the brain stem were selected. Each block was refixed in fresh 15 per cent formalin for a week. The thalamus, caudate, lentiform and putamen were divided in six parts and prepared in semiserial frozen sections of 50 to 70 mμ. Each section was stained by a slight modification of the method of Gros. That is to say, the sections after having been treated with 96 per cent alcohol for half an hour or more were put into 20 per cent silver nitrate,

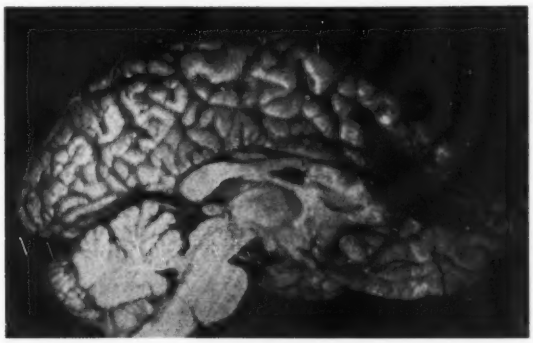


FIG. 1. *Case 1.*

using a quarter or a fifth capsule with a 1 per cent dilution of formalin, before proceeding with the ammoniacal silver nitrate method of Gros. In the same manner controls of normal material of a woman aged 41 years, who died of heart failure and without significant changes in the brain, were prepared of the thalamus and brain stem. According to a previous large experience, this method supplies a uniform staining, and particularly satisfactory is the control of the intensity of impregnation.

For the description of the topography of the cerebral cortex injuries the scheme of Brodmann was used. The sections, which demonstrate the degeneration of the fibers quite well, were drawn by camera lucida and each sketch depicts the alterated interrupted fibers. The "boutons terminaux" are indicated by crossed lines. Each section is accompanied by a corresponding photomicrograph showing details of the degeneration of the fibers or terminations.

DESCRIPTIONS

Case 1. Observation No. 6627. N.C.C., a woman 22 years of age, had had epilepsy with serious mental confusion since childhood. She died 14 days after operation.

Autopsy. The brain was of normal size and weight. A resection of the cortex,

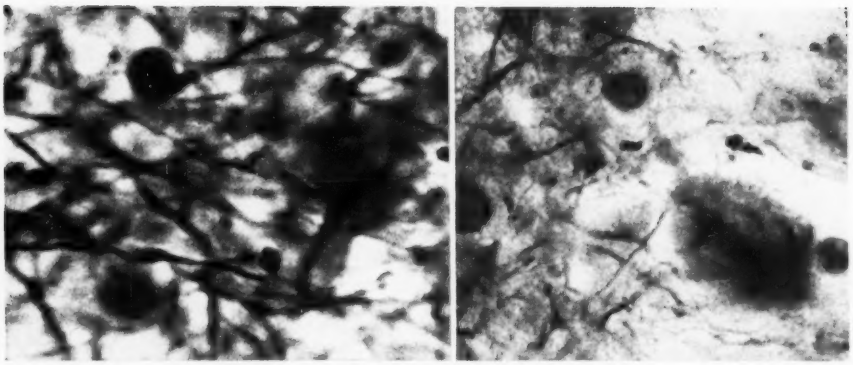


FIG. 2. *Case 1.* Two different sections of mammillary body; one with pathological terminal fibers around nerve cells and the other with hypertrophied "boutons terminaux." Gros method, ×800.

which comprised only a few millimeters of the white matter, can be observed in both hemispheres on the medial surface of the frontal lobe (Fig. 1). This zone corresponds to area 32 and only in a small proportion to areas 9, 10 and 24 of Brodmann. In both hemispheres the lesions were fairly symmetrical.

Secondary degeneration (left hemisphere). Sections made at the level of the anterior third of the thalamus show the small fibers of the medial portion of the internal capsule, presenting degeneration of axons in a narrow part adjacent to the external part of nucleus ventralis anterior thalami. Bundles of hyperargyrophilic fibers are actually seen in the ventral part of internal capsule, passing from here into the nucleus mammillaris medialis in which numerous degenerated terminals can be observed (Fig. 2).

At the same level, fibers from internal capsule entirely degenerated were seen to reach the globus pallidus but it was not possible to establish the final pathway of these fibers because terminal fibers in the control material were abundant and in form of bulb-like structures.

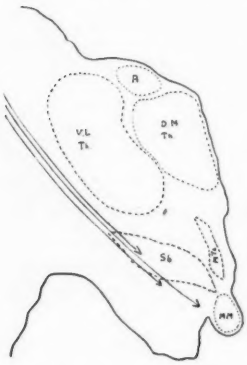


FIG. 3. *Case 1.* Frontal section of thalamus at the caudal level of the mammillary body; the lines with arrows represent degenerated fil. *cs.*

Middle level. The fibers and terminals of the nucleus anterior thalami were normal. The nucleus dorsomedialis and the nucleus submedius did not show degeneration.

From the internal capsule fine fibers could be observed adjacent to the nucleus ventralis lateralis along its lateral part. Some of these passed into the lateral half of the corpus subthalamicum (Fig. 3).

Posterior level. The nucleus dorsomedialis thalami did not show degenerating terminal fibers. The nuclei submedius, parafascicularis, arcuate and centre median had normal terminal plexus. At this level a few fibers enter the corpus subthalamicum, surely from the internal capsule, and end here, in club-shaped fibers around the nerve cells.

The substantia nigra and the peduncle were normal without degenerated fibers.

The changes in the right hemisphere were in general similar to those on the left side.

Case 2. Observation No. 6939. E.N.T., a woman 16 years of age, had presented signs of suprasellar tumor for 1 year. For location and extirpation of the tumor the

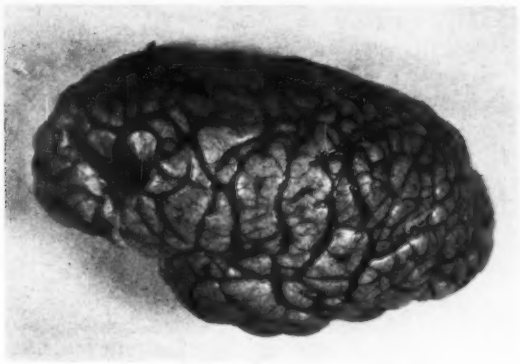


FIG. 4. *Case 2.*

surgeon had to penetrate the left frontal lobe at the level of the medial frontal convolution. The patient died 37 days after operation.

Autopsy. The apparently normal brain presented only a perforating surgical wound in the lateral surface of the left frontal lobe, opposite to the medial frontal

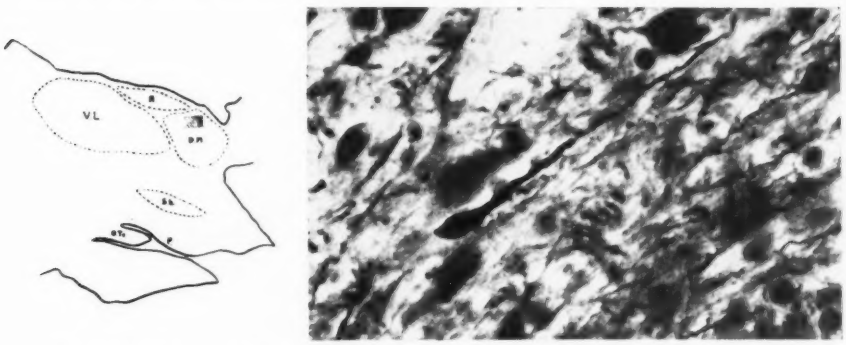


FIG. 5. *Case* 2. Frontal section of thalamus sketched in camera lucida; special marked zone corresponds to photomicrograph of pathological fibers. Gros method, ×800.

convolution at area 46, with a smaller extension at area 10 (Fig. 4). The medial surface of the wound opened into a somewhat haemorrhagic canal at the level of areas 32 and 11 of Brodmann. Autopsy confirmed the presence of a tumor in the body of the sphenoid, which had caused a slight prominence into the subdural space without destroying the dura mater. Rupture of the dura mater, or invasion of the ventral part of the brain could not be demonstrated, either by autopsy or microscopical examination of the region.

FIG. 6. *Case 2.* Frontal section of globus pallidus sketched in camera lucida; special marked zone corresponds to photomicrograph of hypertrophied "boutons terminaux." Gross method, ×800.

Secondary degeneration. Sections at the anterior level of the thalamus show hypertrophic fibers adjacent to the nucleus anterior thalami, but none of these fibers terminate here. In the medial part of the internal capsule there are entirely degenerated hypertrophic moniliform fibers which sweep down into the lateral half of nucleus dorsomedialis thalami and end like retraction masses around the nerve cells (Fig. 5). Other fibers separate from the internal capsule and end entirely degenerated in the nucleus lateralis hypothalami.

The tractus mammillothalamicus, the nuclei mammillaria and the nucleus anterior thalami were normal.

Middle level. Here degenerated fibers from the medial part of the internal capsule end in the lateral and basal part of the nucleus dorsomedialis thalami and in the dorsal and lateral part of nucleus submedius.

The pallidus at this level shows some hypertrophic peridendritic "boutons" (Fig. 6).

Posterior level. Pathological fibers from the ventral part of internal capsule are

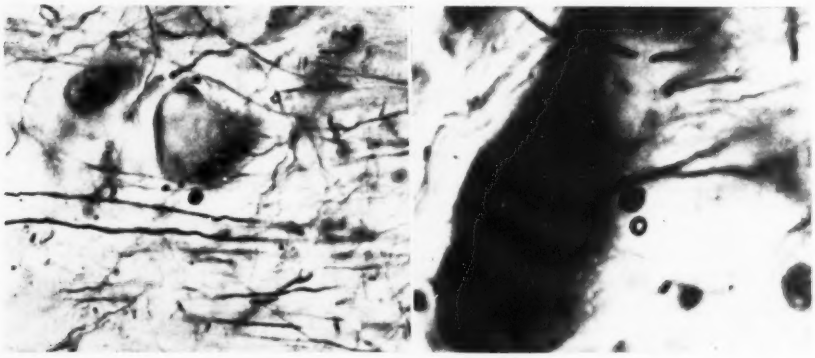


FIG. 7. *Case 2.* Photomicrograph of hypertrophied, club-shaped terminals around nerve cells of corpus subthalamicum. Gros method, ×320 and ×800.

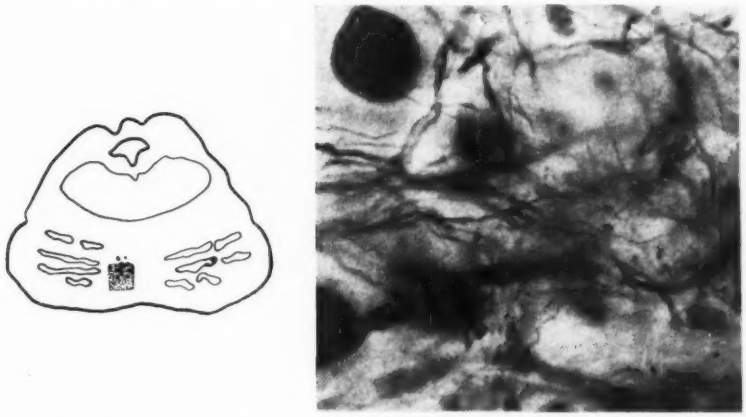


FIG. 8. *Case 2.* Horizontal section of rostral part of the pons, sketched in camera lucida; special marked zone corresponds to photomicrograph showing numerous hypertrophied "boutons terminaux." ×800.

seen to reach the corpus subthalamicum, ending in it in club-shaped terminals (Fig. 7). Other fascicles appear to end in the dorsolateral part of the nucleus ruber. Many fine and degenerated fibers passed through lower centers. Fascicles broken up within the internal capsule were seen adjacent to the lateral part of the nucleus ventralis lateralis, descending to the peduncle bordering the corpus subthalamicum.

Secondary degeneration in the pons. The serial sections of the pons showed pathological fibers ending like hyperthophied pericellular "boutons terminaux," in relation to cells of nuclei pontis adjacent to the midsagittal plane (Fig. 8).

DISCUSSION

The secondary degenerations described in this paper are those that relate to wallerian degeneration. Degenerative lesions of the fibers produced by surgical trauma are not considered here. In this material the degree of direct degeneration provoked by trauma is estimated to be 10 mm. in depth. Vaz Ferreira, in his experimental material, stated that the surgical lesions were 2 mm. in extent.

The interrupted fibers were considered as pathologic when they were swollen, hyperargyrophilic or fragmented, according to Meyer.[9] The "boutons terminaux" were considered to have undergone changes if they were deformed, of large size, and overimpregnated by the ammoniacal nitrate of Gros. Doubtful lesions were excluded, since the use of silver impregnation in a large routine material shows that insignificant morphological changes of the fibers are also to be observed in normal material.

In general, the secondary degeneration is more marked in Case 2, in which the patient survived for 37 days after operation. Meyer, in a study of 4 cases after leucotomy, also found that degeneration of the fibers was more advanced in cases of long survival.

Hypothalamus. In this investigation it was confirmed that in the human

brain there exists a direct efferent frontohypothalamic pathway, which arises in agranular frontal cortex. In fact, there are signs of end degeneration in the mammillary body in Case 1, with topectomy of area 32. In Case 2, with resection of areas 46 and 10, terminal degeneration was found in the nucleus lateralis hypothalami. In this particular case, there was a concomitant lesion of area 32 and the exact relation of each area with the efferent connexions cannot be determined. This confirms the studies of Beck, Meyer and Le Beau.[3] These authors described a direct corticohypothalamic pathway originating anterior to the agranular cortex and ending in the ventromedial and lateral hypothalamic nuclei.

In Case 1, in which the principal lesions included area 32 and small parts of areas 24 and 9, the findings of Meyer[9] were verified. There are signs of terminal degeneration in the mammillary body. Also in Case 1, some fibers definitely ending in the corpus subthalamicum were seen.

It is interesting that Meyer regarded area 24 as origin of the hypothalamic contingent. Beck and co-workers[3] stated "whether this tract (corticohypothalamic) arises from the medial orbital or dorsal frontal cortex cannot be determined from the present material."

In the material presented here, the lesions in Case 1 were confined to the medial prefrontal region and in Case 2 to the prefrontal and partly to medial prefrontal areas. It is possible that the hypothalamic terminal degeneration in these two cases was related to area 24, as stated by Meyer, and also to area 32, according to our material. There have been two previous anatomical reports on the direct connexion between the frontal cortex and the hypothalamus.[3,9]

Thalamus. In Case 1, with extensive interruptions of fibers of area 32 and part of area 24, there were no corticofugal connexions of the frontal lobe with the nucleus anterior thalami. No pathological changes in this nucleus were seen. Ward,[11] in experimental studies, was also unable to find terminal degenerations after ablation of area 24, using the method of Marchi. Bailey and co-workers[1] did not obtain any effect in the nucleus anterior thalami after strichninization of areas 24 and 32.

In complete agreement with the findings of Meyer,[9] in our Case 2 abundant degenerated end fibers were found in the nucleus dorsomedialis thalami and nucleus submedius, related to the first, according to Meyer, Beck and McLardy.[8] It is interesting that Freeman and Watts[6] described an efferent projection of this nucleus to area 46. The results of the aforesaid authors were confirmed in our case with destruction of area 46.

Extrapyramidal nuclei. Some degenerating fascicles of fibers were seen ending in the corpus subthalamicum and in the nucleus ruber in Case 2. In Case 1, it was not possible to determine whether the fibers passing through the pallidus ended here because normal "boutons" varied in size in the controls.

Prefrontopontine bundle. The course and the end of this fascicle has been demonstrated by Beck.[2] In our Case 2 it was possible to follow the course of

the prefrontopontine bundle to the pons. According to Beck and our observations, lesions of the lateral convexity of the prefrontal region produce degeneration of the bundle.

SUMMARY

1. Two cases of frontal leucotomy were studied with the silver impregnation technique of Gros, in order to determine the course and terminations of the corticofugal fibers in the subcortical nucleus.

2. Lesions of the medial surface of the frontal lobe (Case 1) showed degenerated terminal fibers in the nucleus mammillaris medialis.

3. Lesions in the prefrontal area (Case 2) showed degenerated fibers in the nucleus dorsomedialis thalami, nucleus submedius, nucleus lateralis hypothalami and corpus subthalamicum. Also in Case 2 there was degeneration from the prefrontopontine bundle to the pons.

I wish to express my gratitude to Miss G. Vilches for her help in preparing the graphic material.

REFERENCES

1. Bailey, P., v. Bonin, G., Davis, E. W., Garol, H. W., McCulloch, W. S., Roseman, E., and Silveira, A. Functional organization of the medial aspect of the primate cortex. *J. Neurophysiol.*, 1944, *7:* 51–55.
2. Beck, E. The origin, course and termination of the prefronto-pontine tract in the human brain. *Brain*, 1950, *73:* 368–391.
3. Beck, E., Meyer, A., and Le Beau, J. Efferent connexions of the human prefrontal region with reference to fronto-hypothalamic pathways. *J. Neurol. Neurosurg. Psychiat.*, 1951, n.s. *14:* 295–302.
4. Carrea, R. M. E., Reissig, M., and Mettler, F. A. The climbing fibers of the simian and feline cerebellum. Experimental inquiry into their origin by lesions of the inferior olives and deep cerebellar nuclei. *J. comp. Neurol.*, 1947, *87:* 321–365.
5. Clark, W. E. Le G., and Meyer, M. The terminal connexions of the olfactory tract in the rabbit. *Brain*, 1947, *70:* 304–328.
6. Freeman, W., and Watts, J. W. The thalamic projection to the frontal lobe. *Res. Publ. Ass. nerv. ment. Dis.*, 1948, *27:* 200–209.
7. Glees, P. Terminal degeneration within the central nervous system as studied by a new silver method. *J. Neuropath. exp. Neurol.*, 1946, *5:* 54–59.
8. Meyer, A., Beck, E., and McLardy, T. Prefrontal leucotomy: a neuro-anatomical report. *Brain*, 1947, *70:* 18–49.
9. Meyer, M. A study of efferent connexions of the frontal lobe in the human brain after leucotomy. *Brain*, 1949, *72:* 265–296.
10. Vaz Ferreira, A. Silver studies of degenerating thalamo-cortical connections. *J. Neuropath. exp. Neurol.*, 1952, *11:* 44–52.
11. Ward, A. A., Jr. The cingular gyrus: area 24. *J. Neurophysiol.*, 1948, *11:* 13–23.

OCCLUSION OF THE MIDDLE CEREBRAL ARTERY

UNDER NORMOTENSION, AND ANEMICALLY INDUCED AND CHEMICALLY INDUCED HYPOTENSION*

BRUCE RALSTON, M.D.,† THEODORE RASMUSSEN, M.D.,‡
AND THELMA KENNEDY, M.S.

Division of Neurological Surgery, University of Chicago, Chicago, Illinois

(Received for publication September 21, 1954)

DURING the past four years, there has been a growing interest in the use of induced hypotension for surgical procedures. Whereas this was previously accomplished by arteriotomy and retransfusion,[4] most recent work deals with the reduction of blood pressure by means of "neurogenic" or "ganglioplegic" mechanisms such as are available in total spinal anesthesia[5] and by chemical ganglionic blocking agents.

During hypotension induced by ganglionic blocking agents, the blood pressure falls as a result of arteriolar dilatation,[13] the pulse generally remains stable, capillary refill time is unaltered, the skin is pink and respiration is slow. In hypotension produced by bleeding there is associated arteriolar constriction leading to capillary stasis, anoxia and the increased danger of irreversible tissue damage.

The clinical use of ganglionic blocking agents (mostly of the methonium group) has now become extensive, both in the United States and abroad. A number of complications have been reported. Among these are cerebral artery occlusion, decerebration, and blindness.[2,6,7] Other workers, however, have been impressed with the increased safety with which certain major surgical procedures can be carried out with the help of this method.[12,13,18]

Perhaps the most important area for the application of this new method has been in neurological surgery. Its use has markedly reduced the hazard of operating on large vascular meningiomas and some of the major vascular anomalies of the brain. Neurosurgical reports of experiences with blocking agents have been almost uniformly favorable. They indicate that fatalities have been low and that those that have occurred are not generally traceable to the hypotension.[1,12,13,17,18]

Despite the use of methonium compounds in several thousand cases, little has been done experimentally to check on the clinical impressions or to study the altered vascular dynamics or to search for pathological changes in the vascular tree. Morris *et al.*[11] found in unanesthetized humans, in the supine position, a significant decrease in cerebral blood flow following

* This study was supported by research grant B-532 from the National Institute of Neurological Diseases and Blindness, of the National Institutes of Health, Public Health Service.

† Now at the National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

‡ Now at the Montreal Neurological Institute, Montreal, Canada.

marked depression of the blood pressure. This occurred in the presence of diminished cerebral vascular resistance. Cerebral oxygen consumption was only slightly reduced because of a more complete extraction of oxygen from the blood. They concluded that there is apparently little danger of cerebral anoxia in the supine patient with a mean blood pressure of 55 mm. Hg when employing hexamethonium induced hypotension. Finnerty *et al.*[3] obtained similar results for blood flow and found a critical rate of flow at which symptoms appeared. Flow studies by Stone,[6] however, following administration of hexamethonium to young adults, showed no change despite reduction of mean arterial pressure of 40–50 per cent.

The objectives of this study have been to compare and to contrast, in the monkey, the degree of pathological and electrical disturbance of the brain produced by a controlled vascular lesion (permanent occlusion of the middle cerebral artery), under normotensive, anemically induced, and chemically induced hypotensive conditions.

MATERIALS AND METHODS

The middle cerebral artery of the monkey (*Macaca mulatta*) was permanently occluded and divided at its origin in a manner that has been well standardized and described elsewhere.[8] There was some modification of the technique. Instead of an osteoplastic craniotomy, the procedure was carried out through a large subtemporal craniectomy. Although the artery can be satisfactorily exposed and divided through a small bony opening, a bony decompression of at least 3 cm. diameter was found necessary to prevent death of the animal from cerebral edema associated with the infarction.

All manipulations of the skin and temporalis muscle were done with the electrosurgical unit to reduce bleeding to a minimum. For EEG recording, a Type A, 8 channel Offner machine was used. Recording was carried out from both sides of the head during the operation. This was done through phonograph needle electrodes driven into the skull above the bony opening on the operated side, and from subgaleal needle electrodes contralaterally. The disposition of these electrodes was as has been described previously.[8] Records were made at intervals in the postoperative period, employing the subgaleal needle electrodes.

Blood pressure was measured every few minutes through a plastic cannula inserted into the femoral or common iliac artery and connected to a mercury manometer. Patency was constantly maintained by using a slow continuous high pressure drip of normal saline through the system. This was provided by a modified pressure cooker with a needle valve at the outlet. There was no need for anticoagulants. Blood was removed or infused through this cannula when necessary. An additional cannula was placed in a leg vein for the introduction of drugs. The circle of Willis was approached by an overhanging head technique, thus eliminating the need for intravenous sucrose to facilitate exposure.[8] The middle cerebral artery was dissected free from its covering of arachnoid and cut between clips placed as close to its origin as possible.

Three groups of animals were studied:

1) *Normotensive* (5 animals). In this, the control group, the blood pressure was not permitted to fall, blood transfusion or dextran being given, as necessary.

2) *Anemic Hypotensive* (6 animals). In this group, the blood pressure was lowered

by bleeding to 60–80 mm. Hg. This was accomplished over a period of 10–20 minutes in most instances. Following varying periods of time, the blood pressure was returned to above 100 mm. Hg or to normal, by transfusion.

3) *Arfonad Hypotensive* (10 animals). In this group, the drug Arfonad* (RO 2–2222) was given to reduce the blood pressure. The agent, a thiophanium derivative, was chosen in preference to the methonium compounds because its effect is supposed to be more certain and transient. This allows considerable minute-to-minute control by intravenous drip and a prompt return to normal on stopping the infusion, without the use of vasodepressor substances.[14] In our experience, several animals were resistant to the drug, showing a slight drop or none at all under anesthesia. This was true despite the use of doses larger than those recommended[10] and after which some of the animals stopped breathing. An initial dose of 5–10 mg./kg. in 100 cc. was given as an intravenous drip and supplemental doses were given if necessary. In this group the average rate of blood pressure fall was 2.4 mm. Hg/minute.

The operating table was kept level, in order to eliminate the added effect of postural ischemia.

Following the hypotensive period, the blood pressure was returned to 100 mm. Hg or normal by stopping the drug and by giving dextran, neosynephrine, or ephedrine if necessary. The rise in blood pressure was not as prompt or controllable as in the anemic group, and, as a result, the hypotensive period was frequently somewhat prolonged.

The animals were followed in the postoperative period for from 3–21 days. Animals surviving less than 3 days were not included in the series to insure adequate time for evidence of infarction to appear. The maximum survival time permitted was 3 weeks. At death, the position of the clips was verified, the carotid arteries were perfused with formalin, and a week later the brain was sectioned. The gross extent of softening was plotted on standard diagrams and microscopic sections were taken for check.

The shorter survival time in some animals was probably caused by massive infarcts approaching the third ventricle, or by gangrene resulting from ligation of the principal artery to the lower limb. Because of the latter complication, it was felt that no accurate appraisal of the degree of hemiplegia could be made.

RESULTS

The results are discussed under two headings: pathologic and electroencephalographic. The blood pressure fall in mm. of mercury and the percentage fall this represented, as well as the duration of the hypotension is shown in Table 1.

PATHOLOGIC

A) *Normotensive.* The resulting infarcts were similar to those obtained previously,[8] but were slightly less extensive. Typical examples are shown in Fig. 1. Anteriorly, the head of the caudate nucleus was only slightly involved, sometimes spared, and the cortex was involved only in its inferolateral aspect. The damage usually did not extend above the fissure of Sylvius, and at the thalamic level involved the basal ganglia not at all, or

* Supplies of Arfonad were made available by Hoffmann-La Roche Inc., Nutley 10, New Jersey.

TABLE 1

Average mean blood pressure following arterial occlusion and duration before return to normal

Blood Pressure (mm. Hg)	Normotensive	Anemic	Arfonad
100+	# 9–117 mm. #12–116 mm. #11–105 mm. # 1–104 mm. # 2–100 mm.		#33–110 mm.
90–100			#34–94 mm. (25%)— 70 min.
80–90			#38–88 mm. (35%)—100 min. #40–86 mm. (25%)—120 min. #31–80 mm. (24%)— 90 min.
70–80		#13–76 mm. (41%)— 90 min. #20–73 mm. (38%)—110 min. #21–70 mm. (26%)—100 min.	#36–78 mm. (33%)—100 min. #25–78 mm. (41%)— 90 min. #37–73 mm. (37%)—170 min.
60–70		#17–68 mm. (46%)— 90 min. #15–65 mm. (51%)— 80 min. #14–64 mm. (49%)— 70 min.	#24–64 mm. (20%)— 70 min. #23–60 mm. (30%)—130 min.

#=number of experimental animal
mm.=millimeters Hg mean blood pressure
min.=minutes
%=percentage blood pressure fall

only at their peripheral margin. More posteriorly, softening was slight or absent. In one animal (#2) the total area of infarction was only a few millimeters in extent.

B) *Anemic Hypotensive* (Fig. 2). The distribution of the blood pressure responses can be seen in Table 1. The infarcts in 5 of the 6 animals were almost identical in this pressure group. They were characterized by more extensive and more severe softening than in the normotensive group. Anteriorly, there was marked involvement of the entire head of the caudate nucleus and extension of the softening to cover the whole inferior half of the frontal lobe. At the thalamic level, the infarct extended deeply to involve the basal

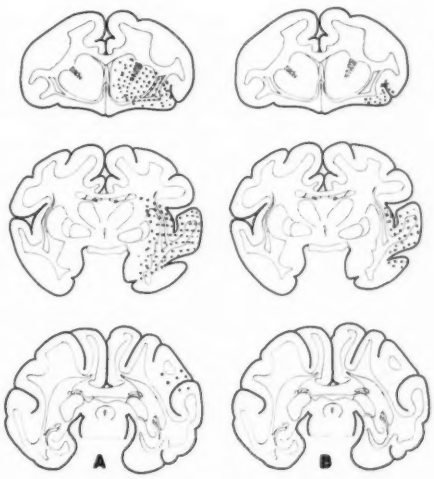


FIG. 1. Diagrammatic representation of area of infarction in normotensive animals. Density of stippling indicates severity of involvement. (A) Monkey #12. (B) Monkey #11.

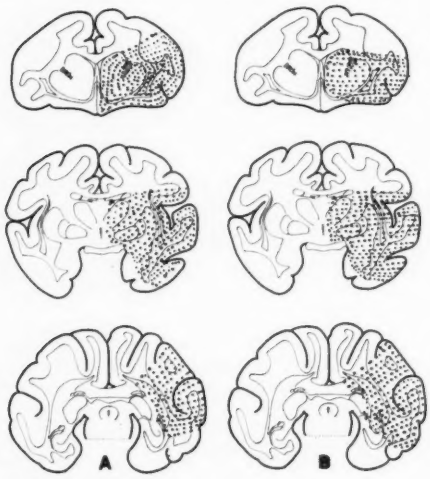


FIG. 2. Diagrams of typical infarcts in anemic hypotensive animals. (A) Monkey #13: 70–80 mm. Hg pressure range. (B) Monkey #15: 60–70 mm. Hg pressure range.

ganglia and the structures in the vicinity of the third ventricle. There was extension of the infarction above the Sylvian fissure and a much larger and deeper involvement posteriorly in the inferior parietal region. One animal (#14) showed no infarction, despite a pressure drop to 64 mm. Hg.

C) *Arfonad Hypotensive* (Fig. 3). As the depressor response could not be so precisely controlled as by exsanguination, the spread of the blood pressure levels was wider and is indicated in Table 1. The areas of involvement were similar to those of the normotensive group but the softening was definitely more marked. The caudate nucleus was softened to about the same degree, but this was present in a greater percentage of animals. Only the inferolateral frontal cortex was involved at this level. At the thalamic level, the infarction did not extend deeply into this structure. There was minimal involvement above the fissure of Sylvius, and posteriorly, the parietal region was only slightly infarcted. The maximum degree of softening was obtained in one animal (#34) in which the blood pressure did not drop appreciably. It should be noted that the variability in extent and severity of the infarction in this group was comparable to that in the normotensive control group.

A direct comparison between the Arfonad and anemic groups can be made in the 60–70 mm. Hg and the 70–80 mm. Hg blood pressure range. In the 70–80 mm. Hg range the percentage blood pressure fall in the anemic group was quite comparable to that in the Arfonad group. The extent and severity of the infarction was clearly less pronounced in the

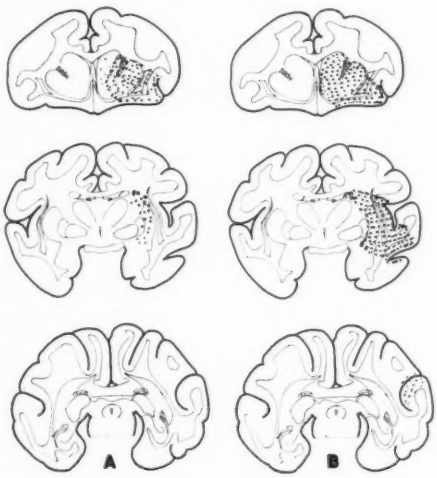


FIG. 3. Diagrams of typical infarcts in Arfonad hypotensive animals. (A) Monkey #36: 70–80 mm. Hg pressure range. (B) Monkey #24: 60–70 mm. Hg pressure range.

Arfonad group, but this in turn was clearly more pronounced than in the normotensive group.

ELECTROENCEPHALOGRAPHIC

The EEG changes were similar to those previously described.[9] The marked lack of correlation between severity of EEG changes and extent of infarction was again noted. Records with minimal or no EEG alteration occurred in each of the three groups as did records with marked loss of normal fast activity, general decrease in amplitude and/or presence of large slow waves.

Seizure discharges were seen in only one of these 21 animals as compared with 10 of 19 monkeys in a previous series.[9] This is of doubtful significance, however, as the total length of recording time in each animal was much less in the present series.

Levels of mean blood pressure as low as 60 mm. Hg in these monkeys did not cause any definite change in the EEG in most of the animals. In the others, the factor of the level of the anesthesia complicates the interpretation. A recent study suggests that the rate of fall of blood pressure is, within limits, more important than the final level in determining EEG changes.[15]

DISCUSSION

The present study confirms the generally held impression[16] that hypotension induced by bleeding increases the brain damage produced by occlusion of a major cerebral artery. In the case of occlusion of the middle cerebral artery of the monkey, hypotensive levels of 70–80 mm. Hg, representing blood pressure falls of 26–41 per cent, produced a maximum middle cerebral artery infarct almost identical to that associated with levels of 60–70 mm. Hg, representing blood pressure falls of 46–51 per cent.

The use of a thiophanium derivative, Arfonad, to produce similar levels of hypotension lessened the size of the infarct but did not completely compensate for the hypotension since the resulting infarcts were somewhat more extensive than in the normotensive animals.

It is of some interest, and a little surprising, that the variability in extent and severity of the infarction in the normotensive and Arfonad groups was similar to that of a previous series in which the blood pressure was not measured.[8]

The mechanism of the protection afforded by Arfonad is not completely clear, despite the previously discussed theorizations, as there is some evidence of a considerable drop in cerebral blood flow in humans rendered hypotensive by hexamethonium, despite decreased vascular resistance. Some observers[11] feel that this may be easily compensated for, at least in the normal. Another possible mechanism may be the blockade of pathways that contribute to vasospasm and focal ischemia.

These results may or may not have any significance as regards injury to the brain produced in other ways, such as that associated with surgical

manipulation. Further experimental investigation of this aspect seems indicated. It should be noted, however, that the benefits of increased safety provided by chemical hypotension may far outweigh the disadvantages of slight increase in local tissue damage. Further efforts to define limits of safety in regard both to blood pressure levels and duration of hypotension would seem advisable.

SUMMARY

1) The extent of cerebral damage following occlusion and division of the middle cerebral artery of the monkey under normotensive, anemically induced, and chemically induced hypotension has been compared and contrasted.

2) Whereas anemically induced hypotension resulted in a marked extension of the infarction, similar depression of the blood pressure using Arfonad, resulted in slight but definite additional damage as compared with the normotensive control group.

3) The characteristic electrical changes of infarction were seen in all groups. The changes were not proportional to the size of the infarction but tended to be less severe in the animals receiving Arfonad, as compared with the anemically induced hypotensive group.

4) It seems unlikely that the EEG will be useful in determining when the depth or duration of chemically induced hypotension begins to be hazardous to the brain.

REFERENCES

1. Anderson, S., and McKissock, W. Controlled hypotension with arfonad in neurosurgery with special reference to vascular lesions. *Lancet.*, 1953, *265:* 754–757.
2. Bromage, P. R. Some electroencephalographic changes associated with induced vascular hypotension. *Proc. roy. Soc. Med.*, 1953, *46:* 919–923.
3. Finnerty, F. A., Jr., Witkin, L., and Fazekas, J. F. Acute hypotension and cerebral hemodynamics. A preliminary report. *Med. Ann. D.C.*, 1953, *22:* 115–116.
4. Gardner, W. J. The control of bleeding during operation by induced hypotension. *J. Amer. med. Ass.*, 1946, *132:* 572–574.
5. Griffiths, H. W. C., and Gillies, J. Thoraco-lumbar splanchnicectomy and sympathectomy. Anaesthetic procedure. *Anaesthesia*, 1948, *3:* 134–146.
6. Hampton, L. J., and Little, D. M., Jr. Complications associated with the use of "controlled hypotension" in anesthesia. *Arch. Surg., Chicago*, 1953, *67:* 549–554.
7. Hampton, L. J., and Little, D. M., Jr. Results of a questionnaire concerning controlled hypotension in anaesthesia. *Lancet*, 1953, *1:* 1299–1300.
8. Harvey, J., and Rasmussen, T. Occlusion of the middle cerebral artery. An experimental study. *Arch. Neurol. Psychiat., Chicago*, 1951, *66:* 20–29.
9. Harvey, J., and Rasmussen, T. Electroencephalographic changes associated with experimental temporary focal cerebral anemia. *Electroenceph. clin. Neurophysiol.*, 1951, *3:* 341–351.
10. McCubbin, J. W., and Page, I. H. Nature of the hypotensive action of a thiophanium derivative (RO 2-2222) in dogs. *J. Pharmacol.*, 1952, *105:* 437–442.
11. Morris, G. C., Jr., Moyer, J. H., Snyder, H. B., and Haynes, B. W., Jr. Vascular dynamics in controlled hypotension: a study of cerebral and renal hemodynamics and blood volume changes. *Ann. Surg.*, 1953, *138:* 706–711.
12. Nicholson, M. J., Sarnoff, S. J., and Crehan, J. P. The intravenous use of a thiophanium derivative, arfonad-RO 2-2222, for the production of flexible and rapidly reversible hypotension during surgery. *Anesthesiology*, 1953, *14:* 215–225.

13. Sadove, M. S., Wyant, G. M., Gittelson, L. A., and Bucy, P. C. Controlled hypotension. *J. Neurosurg.*, 1953, *10:* 272–283.
14. Sadove, M. S., Wyant, G. M., and Gleave, G. Controlled hypotension; a study on arfonad (RO 2-2222). *Anaesthesia*, 1953, *8:* 175–181.
15. Schallek, W., and Walz, D. Effects of drug-induced hypotension on EEG of dog. *Fed. Proc.*, 1954, *13:* 128.
16. Thompson, R. K., and Smith, G. W. Experimental occlusion of the middle cerebral artery during arterial hypotension. *Trans. Amer. neurol. Ass.*, 1951, 203–206.
17. Wiklund, P. E. Controlled hypotension at intracranial operations. *J. Neurosurg.*, 1953, *10:* 617–623.
18. Woodhall, B., Odom, G. L., Stephen, C. R., McClure, C., and Neill, W. R. The use of chemically induced hypotension in the treatment of intracranial aneurysm. *N. Carolina med. J.*, 1953, *14:* 513–516.

INTRACRANIAL HEMATOMAS ASSOCIATED WITH PENETRATING WOUNDS OF THE BRAIN*

JOSEPH C. BARNETT, M.D.,† AND ARNOLD M. MEIROWSKY, M.D.‡

(Received for publication September 29, 1954)

THIS presentation is a study of a consecutive series of 316 penetrating wounds of the brain with particular reference to the occurrence of intracranial hematomas. The data were made available through the concerted effort of the Medical Officers who were for varying lengths of time associated with an Army Mobile Neurosurgical Unit in Korea.[5] This unit was attached to a Mobile Army Surgical Hospital, the most forward medical institution offering major surgical facilities. The period represented is from October 13, 1951 through October 31, 1952. The report is composed entirely of cases in which operation was performed.

By way of definition, any compound wound with entry into the cranial vault and laceration of the meninges, regardless of the severity or extent of cerebral destruction, is considered as a penetrating wound of the brain. This is intended as a broad classification, embracing the through-and-through wounds and other such descriptive terms as tangential wounds of the brain. The conventional and generally accepted terms are used in the classification of the hematomas. The subcortical or intracerebral hematomas in this series refer, in general, to hematomas of the missile canal resulting from laceration of vascular radicles in the path of the missile. When a hematoma was found to occupy the ventricular cavity, out of particular interest, this was classified as an intraventricular hematoma. Direct visualization of the hematoma was the criterion for this diagnosis and in all instances the penetration traversed the ventricular system.

It should be stated that an effort was made to differentiate between a hematoma and a few small clots. It is admittedly difficult to conceive that a missile could penetrate the cerebral substance without provoking some small amount of hemorrhage. Of assistance was the impression gained at operation as to the actual degree of space occupancy. As a rule following sufficient exposure, the hematoma forcefully extruded from its confines. On the contrary, with avulsive wounds of the head and brain occasionally large hematomas were found; however, they were not confined and therefore were not considered as intracranial hematomas in this series. A confined hematoma in excess of 20 cc. was felt to be of clinical significance and was used as a criterion for the diagnosis of hematoma.

* Presented at the annual meeting of the Harvey Cushing Society, Hollywood, Florida, April 24, 1953.

† Emory University, Georgia.

‡ 811 Bennie Dillon Bldg., Nashville, Tennessee.

ANALYSIS OF CASES

Type of Missile. In 286 of the 316 cases the injuries were the result of shell fragments. Shell fragments include fragments from mortars, mines, grenades or artillery shell. There were 18 instances in which the offending missile was the result of small arms fire. In the remaining cases the injuries were the result of blasting mishaps or the exact agent causing the injury was unknown. The actual size varied from a few millimeters in diameter to several centimeters. In the instances of tangential or glancing wounds, frequently there were no intracranial metallic fragments, the indriven cranial bone fragments being accountable for the cerebral penetration.

Site of Injury. In 260 cases of the series (82 per cent), the primary involvement was limited to one cerebral lobe, not taking into consideration involvement below the cortical level. In 44 cases, more than one lobe was involved in the primary injury. This latter group comprised principally the glancing or tangential, the avulsive, and more extensive type of wound. There were 10 cases in the series in which there were two distinct penetrating wounds, and there were 2 instances in which three distinct penetrating wounds were found.

TABLE 1

Incidence of hematomas in 316 cases of penetrating wounds

Site of Hematoma	No. of Hematomas	Incidence (% of series)
Epidural	13	3%
Subdural	77	21%
Intracerebral	87	23%
Intraventricular	2	0.2%

Of some interest was the location of these wounds. There were 149 penetrating wounds that primarily involved the frontal lobes; 94 involved the parietal lobes; 75 involved the temporal lobes; 25 involved the occipital lobes; and 12 primarily involved the cerebellum. In this series, there seems to be a slight predilection for the left-sided injuries. Fifty-one per cent of the wounds primarily involved the left hemisphere, whereas 39 per cent primarily involved the right hemisphere. Four per cent involved the cerebellum and 6 per cent of the injuries were of a midline nature thus producing bilateral primary involvement.

Incidence of Hematomas (Table 1). Of the 316 cases of penetrating wounds, 146 (46.2 per cent) were complicated with confined intracranial hematomas. Thirty-five of these patients presented more than one anatomically significant hematoma at operation. It was noted that the subdural and intracerebral hematomas were frequently associated. Thirty-one of the 35 patients who were found at operation to have more than one anatomically significant hematoma presented an intracerebral as well as a subdural hematoma.

There were 13 cases in which there was a clinically significant epidural hematoma. This represents 3 per cent of the series. Of these 13 cases, 5 were complicated by the presence of either an intracerebral or a subdural hematoma.

There were 77 cases, 21 per cent of the series, in which subdural hematomas were found. Of the 77 cases, 24 were complicated by the presence of either an intracerebral or an epidural hematoma.

There were 87 cases in which intracerebral hematomas were present. This represents 23 per cent of the series. Of the 87 cases, 33 were complicated by accompanying subdural or epidural hematomas.

There were 2 cases of intraventricular hematoma.[8]

It was noted that some relationship existed between the site of penetration and the likelihood of hematoma (Table 2). Of the wounds primarily

TABLE 2

Relationship between location of wound and formation of hematoma

Location of Wound	No. of Wounds	Associated Hematoma
Frontal	149	32%
Temporal	75	54%
Parietal	94	32%
Occipital	25	58%
Cerebellum	12	52%

involving the frontal lobes, 32 per cent were associated with hematomas whereas 54 per cent of wounds primarily involving the temporal lobes were associated with hematomas. Of the wounds involving the parietal lobes, 32 per cent were associated with hematomas. Fifty-eight per cent of the occipital lobe penetrations and 52 per cent of the cerebellar wounds were complicated with hematomas. It is of interest to note that frontal and parietal lobe penetrations are less frequently associated with hematoma formation than penetrating wounds elsewhere.

Time Factor. Early surgical care for the traumatic neurosurgical patient has been emphasized by many previous writers and observers.[2,4,6,7] In consideration of the time factor in this series of patients, it must be borne in mind that the most reliable data concerning the time of injury were those recorded by the Aidman or the Batallion Surgeon when the patient was first administered medical treatment. There is admittedly a margin of error here, of possibly several hours in some instances, when because of exigencies of battle, the wounded had to await medical treatment. However, when the patient enters the medical evacuation chain, immediate disposition is possible. The helicopter evacuation plan of the seriously injured patient has created a new and now well recognized phase of medical evacuation.[5]

Thirty-nine per cent of the patients included in this series were received

for definitive neurosurgical care within 3 hours after first medical aid; 29 per cent were received within 6 hours, and 10 per cent within 8 hours. Thus within a period of 8 hours a total of 78 per cent of the 316 patients were under definitive neurosurgical care. The remaining patients were for the most part from far distant points or were unavoidably detained for tactical reasons.

Neurological Findings. Fifty-one per cent of the series on admission showed signs of neurological deficit. Central pareses, paralysis, visual loss, astereognosis, aphasia, marked sensorial change, as well as signs of acute intracranial distress were noted, followed and recorded. Of the 146 patients who were found at operation to have a significant intracranial hematoma 105, or 71 per cent, presented on admission or shortly thereafter conclusive evidence of neurological deficit. This figure compared to the over-all percentage (51 per cent) possibly is of some significance.

DISCUSSION

There have been many contributions regarding military neurosurgical casualties as to observation and management resulting from World Wars I and II. However, there are remarkably few references to the actual incidence of intracranial hematomas associated with penetrating wounds of the brain. Schorstein[7] in his study of World War II casualties observed that three-quarters of the penetrating craniocerebral injuries presenting a small wound of entry were associated with intracranial hematoma. In this series no real effort was made to classify the size of the wounds, feeling that the important factor, as Schorstein stated, was the intracranial confinement of the hematoma; this being in contradistinction to the avulsive type of wound in which cerebral and meningeal bleeding readily escapes from the wound.

Matson and Wolkin[3] observed that in instances in which the missile was found located near a cortical surface opposite or within the line of trajectory of the missile, there was likelihood of hematoma formation in the region of the missile. There were 15 instances of this particular situation encountered in the series. In 6 of these 15 a significant hematoma was found in the region of the distantly located missile. It is interesting that statistically this would be comparable to the over-all incidence of hematomas for the series if one were to consider the distant surface injury as a distinct penetrating wound.

The time required for the evacuation of the patient to a major surgical facility is important relative to the incidence of hematoma. A total of approximately 200 cases of penetrating wounds of the brain were reviewed, these cases antedating the forward location of the neurosurgical team. During the early phase of the Korean conflict, neurosurgical casualties were evacuated to Japan for definitive care. The usual time required for transportation of the patient was from 24 to 72 hours. In this group of patients there was a 7 per cent incidence of intracranial hematoma. Some months later when it was possible to offer major surgical facilities within Korea, a neurosurgical team was established at the Evacuation Hospital level. The evacuation time at this level was lessened, usually to 12 to 36 hours, and

there was a 24 per cent incidence of hematoma. These figures compared to the 46 per cent incidence of hematoma in the present series wherein the majority of the patients reached a facility for definitive care within 8 hours would seem significant. This time difference may be of value in the explanation of the somewhat divergent observations of previous authors.[1]

The high incidence of confined intracranial hematomas in casualties examined and treated within 8 hours, lends added significance to the availability in a theatre of war of mobile neurosurgical teams at division level.

SUMMARY

1. A series of 316 cases of penetrating wounds of the brain, treated by an Army Mobile Neurosurgical Team within 8 hours after injury, have been analyzed with regard to the occurrence of confined intracranial hematomas.

2. The incidence of confined intracranial hematomas was found to be 46.2 per cent.

3. By way of comparison, confined intracranial hematomas were found in 27 per cent of patients treated 12–36 hours, and in 7 per cent of patients treated 24–72 hours after having been wounded.

4. The high incidence of intracranial hematomas associated with penetrating wounds of the brain necessitates earliest possible definitive neurosurgical treatment.

REFERENCES

1. Craig, W. M. Neurosurgery in World War II. *U.S. Armed Forces med. J.*, 1952, *3:* 1–14.
2. Finlayson, A. I. Penetrating war injuries of brain. Desirability of early definitive surgery. *Bull. U.S. Army med. Dept.*, 1945, *3*, No. 87: 61–69.
3. Matson, D. D., and Wolkin, J. Hematomas associated with penetrating wounds of the brain. *J. Neurosurg.*, 1946, *3:* 46–53.
4. Meirowsky, A. M. Penetrating craniocerebral trauma. Observations in Korean war. *J. Amer. med. Ass.*, 1954, *154:* 666–669.
5. Meirowsky, A. M., and Barnett, J. C. Mobile neurosurgical team. *Ann. Surg.*, 1953, *138:* 178–185.
6. Meirowsky, A. M., and Harsh, G. R., III. Treatment of penetrating craniocerebral trauma. In: *Symposium on Military Medicine, U.S. Army Far East Command*, 1951, 85–88.
7. Schorstein, J. Intracranial haematoma in missile wounds. *Brit. J. Surg.*, 1947, War Surg. Suppl. *1*, 96–111.
8. Wannamaker, G. T. Transventricular wounds of the brain. *J. Neurosurg.*, 1954, *11:* 151–160.

AN ELECTROPHYSIOLOGICAL STUDY OF NERVE REGENERATION IN THE CAT

WILLIAM F. COLLINS, M.D.,* JAMES L. O'LEARY, M.D.,
WILLIAM E. HUNT, M.D.,† AND HENRY G. SCHWARTZ, M.D.

Sections on Neurology and Neurosurgery, Washington University School of Medicine, St. Louis, Missouri

(Received for publication September 30, 1954)

INNUMERABLE anatomical studies reflect an abiding interest in nerve repair. In most respects clinical observations have confirmed and extended the anatomical, but complementary electrophysiological studies are also necessary. There have been few of these. A considerable share of clinical interest centers about the return of sensation, and particularly this aspect of nerve regeneration should be advanced by electronic recording techniques.

Berry, Grundfest and Hinsey[1] and Berry and Hinsey[2] studied the conducted potentials of cut and sutured nerves of the cat *in vitro* using the sciatic, its tibial and peroneal branches, and the saphenous. They examined cat nerves at intervals of 17 to 466 days following suture, and their results indicated a close adherence to the relations that prevail between fiber size and conduction velocity in normal preparations. As regeneration proceeded they observed that conduction velocity showed a definitive relation to elapsed regeneration time, although complete recovery of speed was not obtained even at the longest intervals studied. Erlanger and Schoepfle[3] studied crushed nerves of the cat *in vitro*, principally the phrenic; their results indicated that conduction time from above to different distances below a neuroma was not a linear function under 190 days. This they related to tapering of the regenerating fibers. That result appears to be at variance with a discussion note by Grundfest,[4] but in part at least the difference may have been attributable to variations in the techniques employed.

We wished to compare for the same animal the characteristics of regeneration in cut and sutured and in crushed nerves, and to examine the potentials evoked in the posteroventral thalamic nucleus by volleys excited electrically from opposite-sided regenerating segments. It was believed the latter might have significance because of the frequently drawn analogy between sensory disturbances associated with human thalamic lesions and the altered sensations that occur during the regeneration of nerves into skin areas. The superficial radial nerve of the cat was selected for our purpose because it is principally sensory in function, and the locus of its thalamic potential can be detected readily by a probe electrode directed into the thalamus.[6,7]

* Work conducted under a fellowship granted by the National Foundation for Infantile Paralysis. Now of Western Reserve University School of Medicine, Cleveland, Ohio.

† Now of Ohio State University School of Medicine, Columbus, Ohio.

METHOD

Under sterile conditions (nembutal anesthesia) both superficial radial nerves were exposed in each of 31 cats. One of the pair was crushed at the elbow with a small hemostat, and the other was cut across and sutured with interrupted silk epineural sutures. Following operation the animals were examined at frequent intervals to detect the presence or absence of response to light touch, pin prick and pressure applied over the cutaneous distribution. A response to noxious stimulation was routinely obtained from skin previously innervated by either a cut or a crushed nerve at 15–20 days postoperatively. Since this was too early for regeneration to have proceeded to the skin terminals we attributed the finding to overlap. Only 2 animals of the series showed hyperirritability to touch, and these at 60 and 70 days regeneration.

The experimental procedures were also done under nembutal, the animals being used at planned intervals to provide a chronological series between 20 and 180 days. The head was fixed in a holder and a stereotaxic device was used to lower a steel recording electrode (100μ tip exposure) through exposed cortex to the thalamic locus for the superficial radial nerve. To detect that locus stimulating electrodes were applied to the opposite superficial radial (above the neuroma) before the nerve was freed for recording.

Tripolar electrodes (2 grids and ground) were used for *in vivo* stimulating and recording. These were so arranged that a stimulating cathode (X) situated 25 mm. proximal to the neuroma was separated by 50 mm. from the nearest recording electrode (Y) of another such tripole situated below the neuroma. A third tripole (Z) was situated further distally, its proximal recording electrode being placed 100 mm. below point X. With minor adjustments the combinations available upon the tripoles enabled us to record different stimulus-response combinations between points X, Y and Z, the 3rd electrode of each tripole serving as needed as a ground point. Each series of records was commenced by recording the evoked thalamic response from points X, Y and Z respectively. Thereafter the nerve was cut above X and the remainder of the records were obtained between points upon the nerve. In each instance we attempted to record monophasically, crushing the nerve under the further removed recording electrode and applying KCl to the cut end. A Grass stimulator was used to deliver single shocks of 0.1 or 1.0 msec. duration. Its output was delivered through an isolation transformer, and a Wheatstone bridge was often used to balance capacitance and resistance changes in the nerve.

At the end of each experiment both nerves were removed and fixed in position (15 per cent formalin) upon cardboard with the sites of the X, Y and Z electrodes' positions marked. Later blocks were cut for osmication and sectioning to reveal the fiber distributions at each of the 3 points, and companion unosmicated blocks were sectioned and stained by Bodian's method for myelinated plus nonmyelinated fibers.

OBSERVATIONS

Fig. 1 illustrates pairs of conducted nerve potentials and evoked thalamic responses for crushed and for cut and sutured nerves recorded 20, 30, 90 and 180 days following operation. All nerve records illustrated were obtained with the stimulating point 25 mm. above (X) and the proximal recording electrode 25 mm. below (Y) the neuroma. Thus, each of these averages conduction over normally rapid upper, and lower, more slowly conducting, re-

generating segments of the contained fibers. The thalamic potentials illustrated were responses to stimuli applied 25 mm. below the neuroma. Records representing the status of the nerve immediately below the neuroma were selected for illustration, because at 20 days the fibers of the crushed nerve

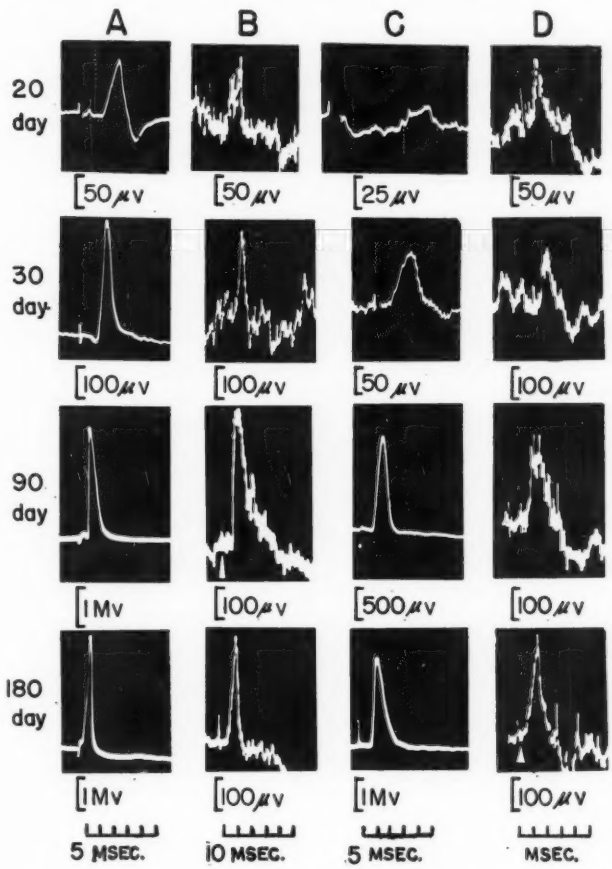


FIG. 1. Pairs of conducted nerve action potentials (A and C) and evoked responses of the postero-ventral thalamic nucleus (B and D) at 20, 30, 90 and 120 days. Columns A and B, crushed interruption; columns C and D, cut and sutured. Comparisons indicate that for latency, duration and amplitude the crushed nerve action potentials were the more advanced for each stage. Compare lower amplitude, longer latency thalamic responses during early as compared with shorter latency, higher amplitude during later regeneration. White triangles indicate shock artifacts in thalamic records.

are just reaching the most distal recording point; those of the cut and sutured nerves had reached it only in the 30-day specimens. Individual variability between different cut nerves recorded after the same time intervals was always greater than amongst the crushed nerves.

The thalamic responses evoked by stimulation of regenerating segments below the interruption differ from those evoked from the nerve proximal to the neuroma in latency and amplitude. The increase in latency to the thalamus was always proportionate to the length and conduction velocity of the regenerating segment, since for a normal nerve it is the largest and fastest of the peripheral fibers that activate the start of the thalamic deflection. The latency from a point on the regenerating segment to the thalamus should be determined by the stage of maturity of the most advanced regenerating fibers. Any significant change in number-size distribution (such as contrasts an early regenerating segment with the normal span above the neuroma) will affect latency and amplitude of the evoked thalamic response. Another obvious factor in reduction in thalamic response amplitude, particularly in

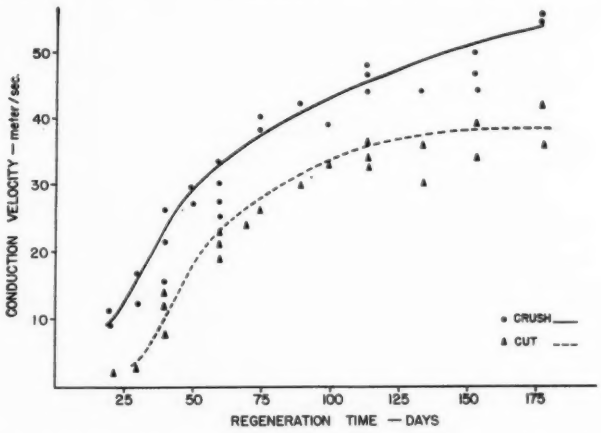


FIG. 2. Plot of conduction velocity/regeneration time based upon average velocity across the neuroma. Note that for earlier regeneration intervals the curves for the crushed and for the cut and sutured nerves are nearly parallel, the crushed ones always showing more advanced regeneration. Similar curves were plotted for a 100 mm. conduction distance and for a 50 mm. stretch entirely below neuroma. These are not illustrated.

early regeneration stages, is the lesser number of peripheral axons in the cross-section of stimulated nerve. As regeneration proceeds the amplitude of the thalamic potential is regained and latency shortens. Always we have found the amplitude of the evoked thalamic response to be proportionate to that of the conducted nerve potential as recorded from the same point. The important consideration, we believe, is that there were no thalamic response alterations that we could not account for in terms of the nerve fiber spectrum and the amplitude of the nerve potential: we did not record after-discharges or increases in amplitude of thalamic potential over the expectancy, either of which could associate with hyperirritability of the conducting pathway.

Fig. 2 summarizes data that concern the increase in fiber velocity with time in cut and sutured and in crushed nerves. The slopes of the two curves

are indicative of the rates of gain in velocity. In the cut and sutured nerves the process is markedly retarded at approximately 120 days. A comparable retardation is noted in the crushed nerves at 90 days but it is of lesser degree. In neither case did the nerves attain at 180 days normal conduction velocity, as determined under our experimental conditions (85–90 meters/sec).

Two sets of data to be discussed later deal with the change in conduction velocity along a regenerating nerve. This is an important consideration in relation to tapering of regenerating fibers. Interpolating our data upon conduction across the neuroma we obtained an approximation of the velocity in the upper 25 mm. of the regenerating segment. This was compared with that through the distal 50 mm. (*Y-Z*). In the crushed nerves to 90 days the

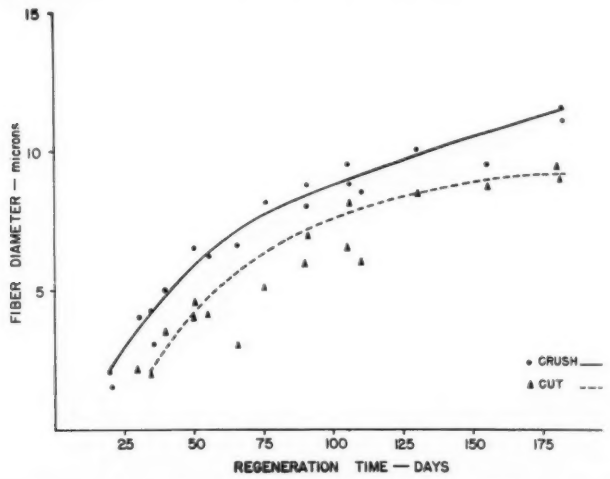


FIG. 3. Plot of outside fiber diameter/regeneration time. Data approximate the characteristics of the conduction velocity curves of Fig. 2.

velocity in the proximal regenerating segment was always greater than in the distal; in the cut nerve this was true to 120 days. Past these times velocities became approximately the same proximally and distally. Maximum fiber-size determinations in the histological material also support these conclusions.

Fig. 3 shows rate of growth in outside diameters of the largest fibers at a point 25 mm. below the neuroma. The slopes of the curves are seen to approximate closely those for conduction velocity/time (Fig. 2). In the crushed nerves the maximum diameter reached was 70 per cent of normal by 180 days, whereas for the cut nerve it was approximately 50 per cent. An overall survey of our histological material also indicates that initiation of propagation precedes myelination. This conclusion is based upon the absence of myelin sheaths in specimens where Bodian stained sections revealed axons, and where conducted nerve potentials had been recorded.

Our threshold values approximated those of the earlier workers, being high in the initial stages of regeneration and reducing with time.

DISCUSSION

Our finding that there is a decrease in conduction velocity and in fiber size as one proceeds distally along a regenerating nerve is consonant with the observations of Erlanger and Schoepfle.[3] At a 31-day interval those authors found the decrease in conduction velocity to be marked with distal progress along a regenerating nerve. In this respect they note a variance with the findings of Grundfest.[4] However, the later publication by Berry, Grundfest and Hinsey[1] does not stress constant velocity, and the curves illustrated are based on regenerating saphenous and tibial nerves at 58 and 90 days respectively with maximum conduction distance of 46 mm. Erlanger and Schoepfle[3] used as much as 160 mm. conduction distance. From our results we would believe that for the conduction distances and regeneration intervals given in the Berry, Grundfest and Hinsey[1] experiments the fibers might have reached a relatively uniform velocity, and a later article by Berry and Hinsey[2] states that at greater conduction distances the conduction velocity is lower. Thus tapering (Young[8]) receives significant support.

We did not make a systematic effort to determine the maximum growth rate of regenerating fibers. However, utilizing the anatomical data of Young[8] that relate to the time required for regenerating fibers to cross the gap in crushed as compared with cut and sutured nerves (5.2 and 7.3 days respectively), our data upon physiological evidence of regeneration approximate those of Young insofar as regeneration past the gap is concerned. We obtained values of 4.0 and 3.4 mm. per day respectively for crushed and for cut and sutured nerves by thus utilizing Young's results.

Prior findings by Hunt and O'Leary[6] and Schricker and O'Leary[7] would indicate that the initial deflection of the evoked thalamic response is activated by the largest fibers of the nerve (*A* spike) after conduction along the dorsal column of the spinal cord. When stimulating below the neuroma we were unable to demonstrate thalamic responses unless there was a conducted action potential. Since conduction velocity in the regenerating segment might be slow, signifying small-diameter fibers, the fact that stimulation evoked a thalamic response indicates that small, slowly conducting regenerating fibers are in continuity through the neuroma with larger more rapidly conducting ones.

The altered latency, duration and amplitude of the thalamic responses evoked from the regenerating segment are evidently determined by nerve conditions, and we found no evidence to indicate hyperirritability of the path central to the neuroma. Two of the 31 animals, in fact, showed during regeneration a marked withdrawal to tactile stimulation in the cutaneous area of the superficial radial nerve, and the evoked thalamic potentials of these were exactly like those of the other animals studied at the same stage.

SUMMARY

Thirty-one animals were studied following crush of one superficial radial nerve and suture of the other. Conducted nerve potentials and evoked responses of the opposite posteroventral nucleus of the thalamus were recorded at stages of nerve regeneration between 20 and 180 days. The salient physiological data of Berry, Grundfest and Hinsey,[1] Berry and Hinsey[2] and Erlanger and Schoepfle[3] are supported, and the results are also consistent with anatomical data provided by Young[8] and Gutmann and Sanders.[5]

Correlation between fiber size and conduction velocity of regenerating fibers was demonstrated, and graphs were constructed to illustrate the rates of increase in both with time. Tapering of regenerating fibers evident in histological preparations correlates with slower distal than proximal conduction velocity to 90 days in the crushed and to 120 days in the cut and sutured nerves. Past these time intervals conduction velocity became approximately uniform over the distances studied. The crushed nerves regenerated at a faster rate than did the cut. While the fibers of neither had reached normal diameter or conduction velocity by 180 days, the crushed nerves were the more nearly normal and appeared to be still increasing at a more rapid rate.

No alterations in latency, duration or amplitude of the evoked thalamic response were obtained that could not be explained upon the basis of the changed physiological properties of the regenerating segment of nerve.

REFERENCES

1. Berry, C. M., Grundfest, H., and Hinsey, J. C. The electrical activity of regenerating nerves in the cat. *J. Neurophysiol.*, 1944, *7:* 103–115.
2. Berry, C. M., and Hinsey, J. C. The recovery of diameter and impulse conduction in regenerating nerve fibers. *Ann. N.Y. Acad. Sci.*, 1946, *47:* 559–574.
3. Erlanger, J., and Schoepfle, G. M. A study of nerve degeneration and regeneration. *Amer. J. Physiol.*, 1946, *147:* 550–581.
4. Grundfest, H. Discussion. *Trans. Amer. Neurol. Ass.*, 1943, 49.
5. Gutmann, E., and Sanders, F. K. Recovery of fibre numbers and diameters in the regeneration of peripheral nerves. *J. Physiol.*, 1943, *101:* 489–518.
6. Hunt, W. E., and O'Leary, J. L. Form of thalamic response evoked by peripheral nerve stimulation. *J. Comp. Neurol.*, 1952, *97:* 491–514.
7. Schricker, J. L., and O'Leary, J. L. Observations on thalamic and cortical responses to peripheral nerve volley. *Electroenceph. clin. Neurophysiol.*, 1953, *5:* 279–290.
8. Young, J. Z. The functional repair of nervous tissue. *Physiol. Rev.*, 1942, *22:* 318–374.

CASE REPORTS AND TECHNICAL NOTES

A CASE OF PERSISTENT CAROTID-BASILAR ANASTOMOSIS ASSOCIATED WITH ANEURYSM OF THE HOMOLATERAL MIDDLE CEREBRAL ARTERY MANIFESTED BY OCULOMOTOR PALSY

FREDERICK MURTAGH, M.D., HERBERT M. STAUFFER, M.D., AND ROBISON D. HARLEY, M.D.

Temple University Medical School and Hospital, Philadelphia, Pennsylvania

(Received for publication August 3, 1954)

To date 20 examples of persistent carotid-basilar anastomosis have been documented in the literature. Sixteen of these were anatomical specimens and 4 others were diagnosed during life by arteriography.[1,2,3] As far as can be determined, none of these examples was associated with aneurysm. Similarly, in no instance was it reported that the anomaly manifested clinical signs and symptoms that could be attributed to the presence of the persistent primitive trigeminal artery into adult life.

The present case constitutes an additional example of the anomaly demonstrated by arteriography and represents, as far as we know, the first report of a case in which the anomaly was associated with aneurysm of the cerebral vessels and an oculomotor palsy was exhibited clinically.

CASE REPORT

The patient, a 62-year-old white male, was admitted on Feb. 25, 1953. Four weeks previously diplopia had developed, followed by ptosis of the left lid. He had no other symptoms except for a mild left supra-orbital pain, which had been present since the development of the diplopia.

Examination. The only objective neurological finding was an external ophthalmoplegia on the left, which involved the lid and the external ocular muscles innervated by the 3rd nerve. The size of the pupils was equal and pupillary activity was normal. Although the patient admitted minimal left supra-orbital pain, there was no hypesthesia in the 1st division of the trigeminal nerve and the corneal reflex was good.

Plain roentgenograms of the skull were originally thought to show no abnormalities.

Left cerebral angiography (Fig. 1) was carried out with the biplane stereoscopic angiographic device, with simultaneous anteroposterior and lateral projections, using a single injection of 10 cc. of 30 per cent Urokon. The films in each plane are obtained at intervals of 0.6 sec.

No abnormality was observed in the capillary or venous phases. The arterial phase demonstrated a somewhat enlarged internal carotid artery, with filling of both anterior cerebral arteries from the left side. There was minimal opacification of the right middle cerebral artery and good normal visualization of the left middle cerebral artery. A large anterior temporal branch of the left middle cerebral ran caudally approximately along the line of the lateral margin of the superior orbital fissure. At a distance of 1 cm. from the origin of this large anterior temporal branch there was an irregular saccular dilatation of the vessel extending laterally for a distance of about 2 cm. along the greater wing of the sphenoid. The other branches of the left middle cerebral artery did not appear unusual.

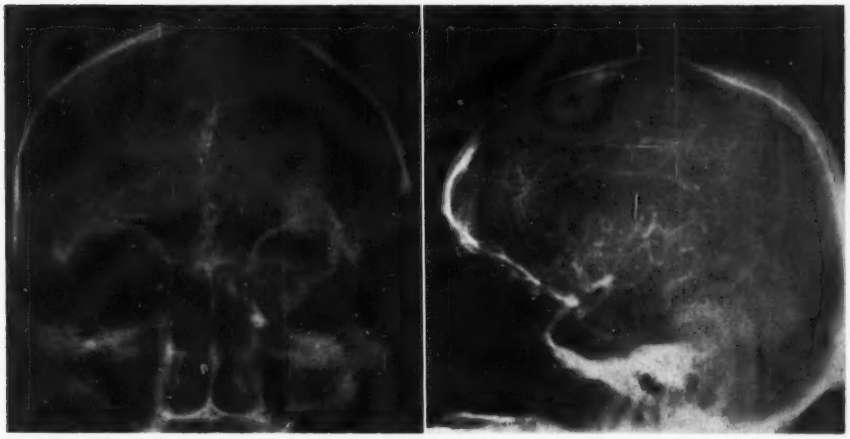


FIG. 1. Left cerebral angiography made with single injection of 10 cc. 30 per cent Urokon and the biplane stereoscopic angiographic device. AP view on left demonstrates the aneurysm situated on the anterior temporal branch of the left middle cerebral artery. In the lateral view the anomalous carotid-basilar anastomosis can be seen.

Following angiographic demonstration of the aneurysm, the plain films were reviewed (Fig. 2) and a small curvilinear calcific deposit was observed at the apex of the saccular portion of the aneurysm, probably representing calcification in the wall of the vessel, although the possibility of slight bone condensation could not be excluded. [This small calcific deposit was still present in films made after surgical treatment of the aneurysm.]

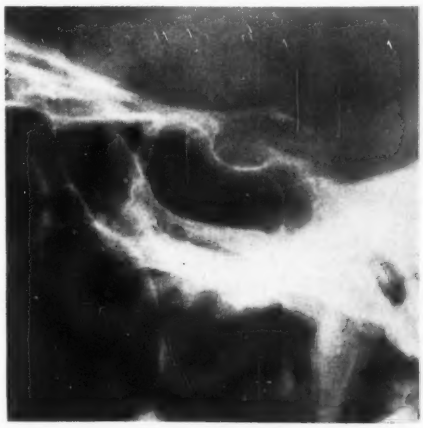


FIG. 2. Plain film of skull—lateral view. Arrow indicates small curvilinear calcification, probably representing calcification in the wall of the tip of the aneurysm.

The basilar artery and both posterior cerebral arteries were opacified, the basilar artery apparently taking its origin from a large anastomotic branch passing through or adjacent to the left margin of the basisphenoid just below the level of the dorsum sellae. The basilar artery measured about 2 mm. in diameter and the anastomotic branch measured about 3 mm., in comparison to the cavernous sinus portion of the internal carotid artery which measured approximately 5 mm. in diameter. The basilar artery and the anomalous anastomosis were well visualized in the stereoscopic lateral projections, but could not be recognized in the anteroposterior projections, apparently because in the films obtained the bony structures and the anterior carotid branches were superimposed on the other opaque midline structures.

The anteroposterior projections showed suggestive evidence of a small branch running medially from the cavernous sinus portion of the internal carotid artery. This branch would correspond to that illustrated by Harrison and Luttrell[1] in their Fig. 5. However, actually this

portion of the carotid artery is proximal to its bifurcation and is supraclinoid in location and therefore could not represent the origin of the anastomosis. The vessel that Harrison and Luttrell have indicated in their figure is apparently not actually the anastomosis. The upper margin of the frontal sinus is often confusing in this location in the absence of stereoscopic films.

The most logical treatment in our case seemed to be a direct surgical attack upon the aneurysm, even though the patient was a right-handed individual.

Operation. A left transfrontal craniotomy was performed on March 6, 1953. With the aid of continuous spinal drainage, the left frontal lobe was retracted with little difficulty and the aneurysm was exposed. It was situated in the tip of the temporal lobe. The tip of the aneurysm was adherent to the dura mater on a 1 mm. base at a point lateral to the superior orbital fissure. This attachment was easily broken, and the aneurysm was completely isolated. The wall of the aneurysm itself was sclerotic and a marked thrust was present with each cardiac systole. The proximal and distal ends of the aneurysm were isolated and found to be composed of soft nonsclerotic vessel. The aneurysm was trapped by placing a stainless steel clip on the anterior temporal branch proximal and distal to the aneurysm.

Course. The immediate postoperative phase was uncomplicated. Approximately 3 hours after surgery, movement of the extraocular muscles innervated by the 3rd nerve on the left was noted to be improved. On the 5th postoperative day an expressive aphasia developed, unassociated with a change in the vital signs, sensorium or other neurological deficit. Forty-eight hours later he began to have Jacksonian convulsions involving the right side of the face, the right shoulder, and the right arm. Again, there was no associated impairment of the sensorium, change of vital signs, or increase in intracranial pressure to suggest intracranial hemorrhage. The seizures became continuous and the patient was given large doses of phenobarbital. Thereafter the seizures were reduced to twitching of the right side of the face only. This continued for 24 hours and then gradually subsided over a period of 3 days. At the end of the 3rd day the seizures ceased and his speech was restored within 48 hours. On the 14th day he was discharged from the hospital ambulatory, with complete recovery from the ophthalmoplegia, free of supra-orbital pain, and with no sign of aphasia except for an occasional transposition of words.

At the date of writing, 18 months postoperatively, the patient's condition remains good with no headache, no diplopia, and a completely normal speech.

DISCUSSION

It is our opinion that the aneurysm in this case is of congenital origin and represents, with the persistent carotid-basilar anastomosis, an example of congenital maldevelopment of the cerebral vascular system. This opinion is based on the fact that morphologically the aneurysm is a saccular one; it is situated on a branch of the middle cerebral artery away from the circle of Willis; and, while the wall of the aneurysm itself was sclerotic, the contiguous vessel on each side appeared grossly nonsclerotic. Clinically, this case did not follow the usual pattern of cerebral aneurysm in that the initial complaint of diplopia followed by ptosis was unaccompanied by the characteristic severe pain one usually experiences with aneurysm.

The pupils of both eyes were equal and the pupillary responses of the left eye were normal. This observation is of interest since Walsh[4] has commented that the "reaction to light is almost invariably abolished or is sluggish."

The possible mechanism by which this group of anomalous structures produced an oculomotor palsy is controversial. It was at first supposed that the 3rd cranial nerve was being compressed by the aneurysm itself at the superior orbital fissure, but on close inspection of the structures running through the superior orbital fissure, it seems unlikely that the aneurysm could selectively compress the somatic motor fibers of the oculomotor nerve without involving the pupillary constrictor portion

or the abducens nerve. In addition, at operation, the aneurysm was found to be fixed to the dura mater at a point lateral to the lateral border of the fissure. Harrison and Luttrell have mentioned the close relationship of the aberrant vessel to the oculomotor nerve at the anterior clinoid process. It is conceivable that fixation of the aneurysm produced traction upon the middle cerebral and internal carotid vessels, which in turn compressed the oculomotor nerve against the anterior clinoid. This premise seems tenable in view of the fact that trapping the aneurysm and releasing it from its point of fixation produced prompt and lasting recovery from the ophthalmoplegia.

Sunderland and Sutton[2,3] both commented on the proximity of the anastomosis to the sensory root of the 5th nerve, particularly the upper portion. Harrison and Luttrell also mentioned this but in none of their cases was there a record of dysfunction of the trigeminal nerve. Left supra-orbital pain was present in this case, but it was minimal and was not characteristic of aneurysm. Since there were no objective signs of dysfunction of the trigeminal nerve, it cannot be definitely established that this represented pressure upon the sensory root or 1st division of the nerve. However, the patient denied pain in the area postoperatively.

SUMMARY

1. A case is presented of persistent carotid-basilar anastomosis associated with saccular aneurysm of the homolateral middle cerebral artery with an oculomotor palsy as the presenting sign. No record of a similar case has been found in the literature.

2. Intracranial clipping of the aneurysm and the subsequent relief of symptoms are discussed.

3. The possible mechanisms producing the oculomotor palsy and left supra-orbital pain are debated.

REFERENCES

1. Harrison, C. R., and Luttrell, C. Persistent carotid-basilar anastomosis. Three arteriographically demonstrated cases with one anatomical specimen. *J. Neurosurg.*, 1953, *10:* 205–215.
2. Sunderland, S. An anomalous anastomosis between the internal carotid and basilar arteries. *Aust. N.Z. J. Surg.*, 1941, *11:* 140–142.
3. Sutton, D. Anomalous carotid-basilar anastomosis. *Brit. J. Radiol.*, 1950, n.s. *23:* 617–619.
4. Walsh, F. B. Clinical neuro-ophthalmology. *Baltimore: Williams & Wilkins Co.*, 1947, xx, 1532 pp.

PTERIONAL MENINGIOMA "EN PLAQUE"

REPORT OF A CASE OF THIRTY-SIX YEARS' DURATION

KENNETH H. ABBOTT, M.D.,* AND BERNARD GLASS, M.D.

Department of Surgery, Division of Neurosurgery, College of Medicine, Ohio State University Hospitals, Columbus, Ohio

(Received for publication August 30, 1954)

With the rapid increase in the numbers of neurosurgeons and neurosurgical operations performed, the patient who has harbored an intracranial tumor for many years is indeed becoming a stranger in our midst. To the pioneers in this field such a patient was not a rarity but today's younger generation of neurosurgeons seldom encounters one. It may be worth while, therefore, to report a single case in which the tumor is known to have been present some 36 years and in which the diagnosis was made by a noted general surgeon 30 years prior to its removal. As may rightfully be suspected, the tumor was a meningioma arising from the lesser wing of the sphenoid with extensive hyperostosis of this bone and associated walls of the orbit.

REPORT OF CASE

The patient was a 59-year-old female referred to us on June 16, 1952 because of a right proptosis, ophthalmoplegia, progressive loss of vision (OD) and right facial disfigurement.

History. About 1916 she began to notice a slight right proptosis, but her family physician did not think enough of this to advise consultation until 1922, when she was referred to Dr. George Crile, Sr. of Cleveland, Ohio with a diagnosis of hyperthyroidism.

The patient relates that she was sent home with a diagnosis of "a tumor behind the eye," and that an operation was not indicated because "it would surely kill me." The exact context of this conversation, of course, was not recorded. However, through the courtesy of Dr. W. James Gardner, we have obtained a copy of the report sent to the referring physician on June 19, 1922:

Dear Doctor C.

Your patient, Mrs. V.B., came in a few days ago and upon examination we find she has a unilateral exophthalmos. I had a suspicion that it might be due to something in the cranium and accordingly she was referred to the Medical Department and X-ray Department and an osteoma was found to be present.

The osteoma is benign and I think it would be just as well to leave the matter for observation. Of course, this picture resembles hyperthyroidism with unilateral exophthalmos. However, the demonstration of the osteoma has put the other construction upon it. Under the circumstances it would seem best to leave both the hyperthyroidism and the osteoma for further consideration.

(signed) G. W. Crile, Sr., M.D.

Fearing this too serious for surgical intervention, nothing further was done about it until June 1952. During these 30 years the proptosis became very slowly more pronounced, but she did not notice any serious loss of vision in the right eye until the summer of 1951, and during the following spring a slight visual loss was noted in the left eye. By the spring of 1952 severe and painful proptosis of the right eye was present with total unilateral ophthalmoplegia (Fig. 1). In the 3 months previous to our examination, a left hemiparesis had slowly de-

* 350 East Broad St., Columbus 15, Ohio.

veloped with "secondary arthritic" involvement of the joints of the extremities (upper more than lower) and Dupuytren's contractures in the left hand became evident. Her daughter reported that the patient's "memory had been fading for a year" with marked personality changes for 8 months.

Examination. The patient's mental processes were somewhat dulled. She displayed "senile irrascibility" and complained constantly and bitterly of pains in her head and right eye.

X-rays of the skull (Fig. 2) disclosed an enormous hyperostosis involving the lesser wing of the right sphenoid, the orbital and cribriform plates, the pterional region and even the zygoma, maxilla and petrous bone. Right carotid angiography (Fig. 3) aided in determining the size of the tumor mass and in locating the origin of its major blood supply.

FIG. 1. Preoperative photograph showing marked proptosis in patient with pterional meningioma of 36 years' duration.

Operation. The tumor mass was of moderate size compared to the many years it had been growing, measuring in its AP diameter approximately 7 cm. inferiorly (along the floor of the middle fossa) to about 3 cm. or 3.5 cm. superiorly. Medially, it extended over the tuberculum sellae beneath the chiasm to the left of the midline. The lesser wing of the sphenoid and the orbital plate were enormously thickened. The combined mass of the "free" tumor and the hyperostosed bone was of no little size and consequently had caused considerable atrophy of the right frontal lobe. The "free" tumor mass was of very soft consistency, allow-

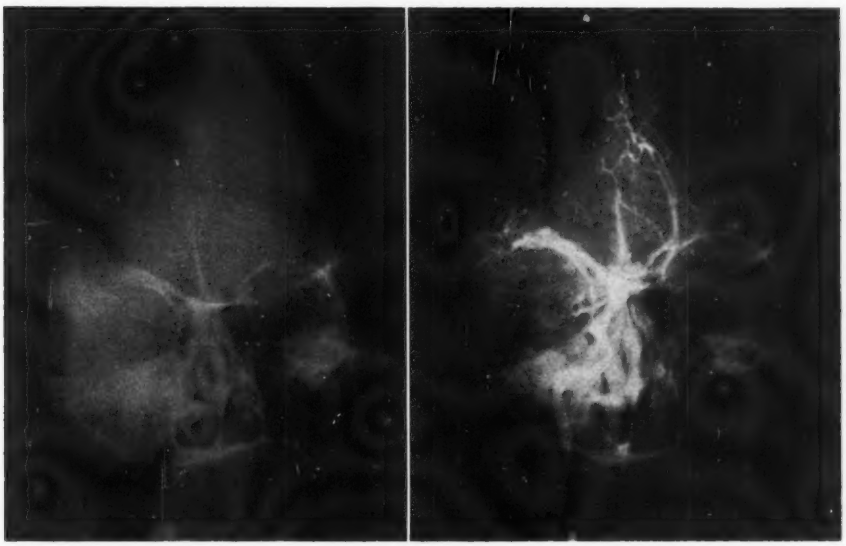


FIG. 2. (*left*) Posterior-anterior roentgenogram disclosing severe right-sided hyperostosis of the lesser wing of the sphenoid, pterion, orbital roof, maxilla and petrous bone.

FIG. 3. (*right*) Anterior-posterior cerebral angiogram disclosing marked distortion of the carotid siphon, and middle and anterior cerebral arteries by the tumor.

ing the removal of its major portion by suction. In order to decompress the orbit the lateral three-quarters of the lesser wing of the sphenoid, including the sphenoidal ridge, the roof and lateral walls of the orbit and the major portion of the bone forming the pterion were rongeured away. This proved to be a very time-consuming but only moderately bloody procedure. A fair amount of tumor tissue was present within the orbit, infiltrating the lateral orbital muscles; this was carefully dissected away. The optic nerve was exposed and well decompressed, and it was observed that the optic canal was some 3.2 cm. long.

Histological Report. The tumor proved to be a mixed type of meningioma (meningotheliomatous and fibrous) with marked invasion of bone but without any sarcomatous characteristics.

Course. The operative procedure was well tolerated, save for a temporary exacerbation of the psychotic behaviour and the left hemiplegia. She recovered slowly from both the psychosis and the hemiplegia and now cares for her "aged and ailing husband." A very mild proptosis persists with complete ptosis of the upper eyelid. The eye turns up and out 15 degrees; there is complete palsy of all the extraocular muscles except the external and superior rectus muscles. The visual acuity has improved; it is now 5/15 O.D. She no longer experiences the head or eye pains.

Comment. This case was of particular interest because of (1) the 36 years or more it undoubtedly had been growing; (2) the extreme degree of hyperostosis; (3) the involvement of not only the walls (posterior, superior and lateral) of the orbit, but also the zygoma, maxilla and the roentgen evidence of hyperostosis of the petrous portion of the right temporal bone, and (4) the amount of "free" tumor present was surprisingly large for this type of growth, which probably should be classified[2] as a pterional meningioma "en plaque" (with considerable soft tumor formation).

DISCUSSION

When one reviews the Cushing and Eisenhardt[2] classic monograph on the meningiomas, it becomes apparent that meningiomas may be present for many years. They cited not only their own cases but many from the literature in which there was an associated hyperostosis of long duration. Certainly in reading Cruveilhier's famous *Anatomie Pathologique*[1] it would appear that he recognized their long duration as well as their probable origin from the arachnoid. Cushing and Eisenhardt reported sphenoidal ridge tumors with known symptoms for 14 years (inner ridge), 21 years (middle ridge) and 13 years (outer ridge—"en plaque"), the average duration of symptoms in the pterional "en plaque" group being about 7 years. In the case herein reported the patient appears to have had her tumor much longer than any of these and it may be one of the longest on record, at least since histologic diagnoses have become reasonably dependable and neurosurgeons so plentiful.

SUMMARY

The case of a 59-year-old female who had a pterional meningioma of at least 36 years' duration is reported. The tumor invaded the orbital walls as well as the orbit and extended back from the sphenoidal ridge into the middle fossa to the petrous bone. Successful removal of the major portion of the tumor with extensive decompression of the orbit has markedly improved the patient's general condition and reduced the proptosis.

REFERENCES

1. Cruveilhier, J. Anatomie pathologique du corps humain. *Paris: J. B. Baillière*, 1829–1835, *1:* liv. 8, pl. 2, fig. 2.
2. Cushing, H., and Eisenhardt, L. Meningiomas. Their classification, regional behaviour, life history, and surgical end results. *Springfield, Ill.: Charles C Thomas*, 1938, xiv, 785 pp.

SOLITARY *ASPERGILLUS* GRANULOMA OF THE BRAIN

REPORT OF 2 CASES

IRA J. JACKSON, M.D., KENNETH EARLE, M.D., AND JOSE KURI, M.D.
Division of Neurological Surgery, Department of Surgery, and Department of Pathology, University of Texas and John Sealy Hospital, Galveston, Texas

(Received for publication September 3, 1954)

Fungous infections of the central nervous system caused by *Aspergillus* are quite unusual. We were able to find a dozen reports of these lesions in the literature. However, only 7 occurred as solitary intracranial granulomatous tumors. Therefore we believe that the addition of 2 such cases of a single space-occupying mass may be of interest.

Aspergillus is a microscopic fungus consisting of nonseptate, elongate hyphae which originate in mycelial foot cells and bear conidiophores of characteristic structure. There are over 350 authenticated strains.[10,15] The fungus is a regular inhabitant of the soil, occurring predominantly in warm damp climates. It is frequently isolated from cereal products, unmilled grain, hay, and other stock feeds. The fungus is generally found as a laboratory contaminant. *Aspergillus fumigatus* is perhaps the most common species producing disease in man but it is likely that this situation results from factors other than its being most pathogenic, such as frequency of effective exposure.

The infection usually involves the lungs, skin, external ears, paranasal sinuses, orbit, and occasionally the bone and meninges.[4] The common pathological picture is one of necrosis and acute inflammation.[1] However, the central nervous system is not only a rare site for this fungus, but also may present the pathological features of chronicity.

In 1897 Rénon[14] observed pulmonary *Aspergillus* as an occupational disease in wig makers and pigeon feeders. It was not until almost 50 years later that Coe[3] reported such a case which was brought to trial before the industrial accident board and judged to be an occupational disease. The literature reveals in general two different types of central nervous system affliction. The commoner is that of a systemic infection with cerebral metastasis, and the other is intracranial lesions with or without an adjacent portal of entry. Moniz and Loff[11] in 1931 reported the necropsy findings of a left frontal lobe abscess in which the etiological agent was not cultured but was morphologically compatible with *Aspergillus*. Since the patient had a history of a corneal ulcer, the authors postulated that the orbit was the portal of entry for the fungus. In the same year Just[9] recorded a case of right frontal lobe abscess and granuloma in which the *Aspergillus* fungus was cultured from the aspirated surgical fluid. At autopsy, only the frontal lobe lesion was present and the surgeon felt that the mode of entrance intracranially was via the nose and frontal sinus. Several years later Guillian *et al.*[7] presented the necropsy findings of a patient with a right frontal lobe abscess and localized basilar meningitis which appeared to have been caused by *Aspergillus* although no culture was obtained. They, too, were of the opinion the fungus entered through the orbit. Oppe[12] as early as 1897 reported a case of *Aspergillus* of the brain in which the primary infection was in the sphenoid sinuses. Wät-

jen[16] in 1928 told of such a cerebral extension with the primary infection arising in the ethmoid cells.

We were able to find only two reports, one being questionable, in which the patient benefited by surgical intervention. Peet[13] presented before the American Neurological Association in 1946 the case of a $7\frac{1}{2}$ year-old child who did quite well following the removal of a large dense fibrotic mass containing many small abscesses from the cerebellum. A pure culture of *Aspergillus* was obtained. There is no note as to the length of the followup. However, Cawley's[2] report a year later from the same institution describes the case study of a patient which appears to be the same that Peet had presented. This patient died 9 months after admission to the hospital despite intensive and protracted treatment, including surgical intervention, with a downhill course. The other possible surgical success was reported by David *et al.*[5] in 1951 in which an encapsulated temporal lobe abscess was removed containing *Aspergillus amstelodami*. Their patient did well following surgery, but there was no notation as to the length of the followup.

Cawley's account in 1947 was probably the first report of a case of generalized *Aspergillus* infection with involvement of the central nervous system. He reported another such situation several years later, but the pathological studies are not too convincing regarding the central nervous system involvement.[6] Several other reports in the literature deal mainly with *Aspergillus* meningitis.[8]

CASE REPORTS

Case 1. JSH #88041. S.D., a 36-year-old colored male, was admitted on July 10, 1953 to the Neurosurgical Service of the John Sealy Hospital because of visual loss. About 16 months previously he began to have some frontal headaches. A month before admission a rapid loss in visual acuity occurred.

He had come to the Out-Patient Clinic a year before complaining of "sinus trouble" of about 8 years' duration. The otolaryngologists felt the patient had a chronic sinusitis and nasal polyps. They removed the nasal polyps and lavaged his antrums. A considerable amount of pus was present on the right but none on the left side. The right side continued to drain for several months. He did not return to the Out-Patient Clinic until almost a year later when he presented himself because of failing vision.

He stated that 15 years previously he had been struck over the right eye and since then had no visual perception in that eye.

Examination. He was an afebrile, well developed colored male who did not appear acutely or chronically ill. The neuropsychiatrists felt that he exhibited no abnormal mental signs. His sense of smell was absent bilaterally. There was no light perception nor reaction to light in the right eye. Vision in the left eye was 3/200. Visual fields revealed a concentric constriction of the left field. There was optic atrophy on the left. The right fundus was not visualized as there was a partial dislocation of the right lens with capsular opacity.

Roentgenograms of the skull showed slight separation of the coronal sutures, erosion of the right petrous ridge, and a calcified pineal gland displaced posteriorly and down. X-rays of the chest, urinalysis, and blood and spinal serology revealed no abnormalities. Findings on peripheral blood studies were normal except the eosinophilic count ranged between 7 and 24 per cent on several occasions. The spinal fluid pressure was 280 mm. of water with 288 WBC/c.mm., 143 mg. per cent of protein and a Lange curve of 00000000.

A bilateral carotid arteriogram was done on July 14, 1953 which revealed a large expanding lesion in the right anterior fossa (Fig. 1A).

Ten days later a pneumoencephalogram demonstrated a definite displacement to the left of the lateral and third ventricles (Fig. 1B).

Operation. A right frontal craniotomy (I.J.J.) was done on July 24, 1953 under general anesthesia. The entire frontal lobe was almost completely replaced by a hard mass of tumor

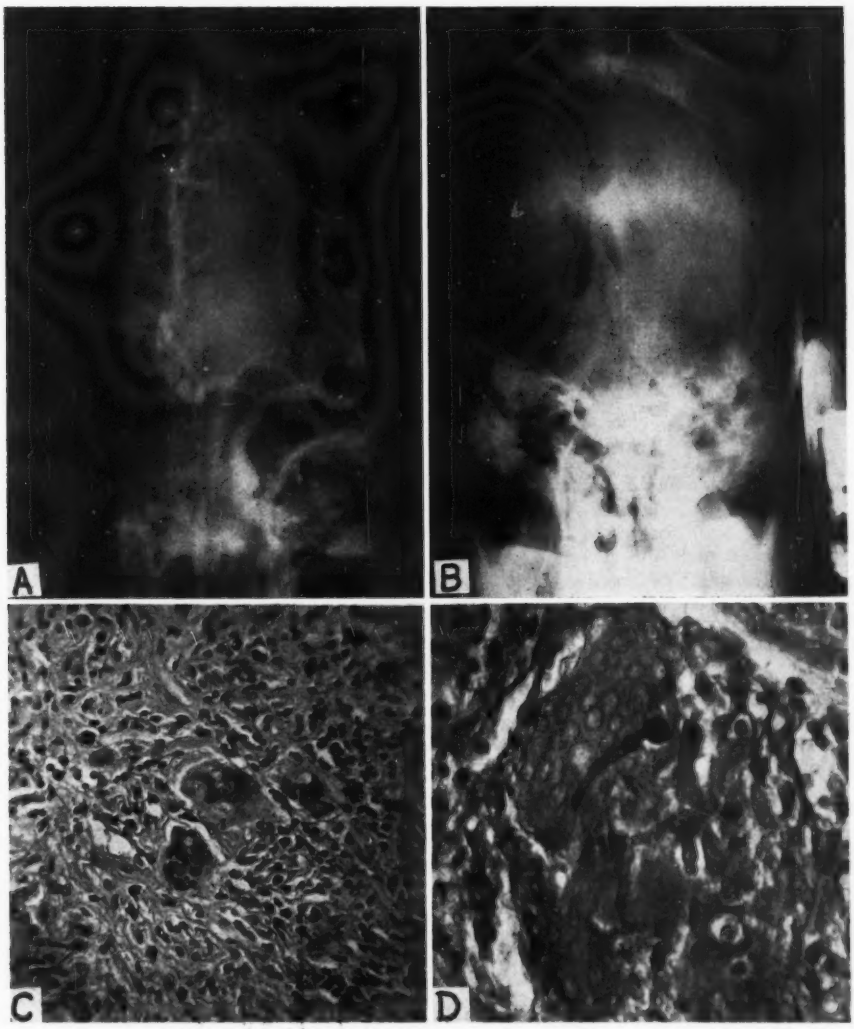


FIG. 1. *Case* 1. (A) Arteriogram showing lateral shift of anterior cerebral arteries. (B) Pneumoencephalogram showing shift of ventricular system from right to left. (C) Microphotograph of granuloma showing foreign body giant cells, fibroblasts, epithelioid cells, mononuclear cells and collagenous stroma. Hematoxylin and eosin stain, ×85. Organisms appear as clear spaces in this stain. (D) Microphotograph of granuloma. Periodic acid-Schiff stain showing septate fungus is morphologically compatible with *Aspergillus*.

tissue leaving only a rim of frontal cortex. The tumor extended over the entire floor of the anterior fossa and posteriorly to the chiasmal region and sphenoid ridge. It had grown over to the left side of the chiasm. This mass was so hard that it was even difficult to cut with a

cautery knife. It was exceptionally avascular but firmly adherent to the floor of the anterior fossa. In no area of the removal was there any softening, discoloration, or cyst formation. The entire frontal lobe with a great part of the mass was removed, leaving the attachment to the floor of the anterior fossa intact.

Postoperative Course. The patient's course was progressively downhill. He expired on Sept. 12, 1953, about 7 weeks after operation. During the postoperative period a multitude of laboratory studies were carried out. Skin tests for blastomycosis, coccidioidomycosis and histoplasmosis were negative. Tuberculin skin test was negative at a dilution of 1:1000 but positive at 1:100 after 48 hours. During the very early postoperative period an infection developed beneath the operative skin flap from which *Staphylococcus aureus* was cultured. This organism was also obtained from the spinal fluid on three occasions. His temperature ranged for the most part between 99° and 101°. There were only several occasions on which it went to 102°. He was given antibiotics to no avail. A great many lumbar punctures were done and the spinal fluid continued to show several hundred white cells and an elevated protein of over 100 mg. per cent. The Lange test exhibited a meningitic curve and on two occasions the Kolmer test was positive. Repeated examinations of the fluid failed to reveal acid-fast organisms and fungi. His demise was attended with a pulmonary embolus.

Pathological Findings. (1) *Surgical* (S-41130). The surgical specimen, weighing 10 gm., consisted of multiple irregular pieces of rubbery gray tissue and softer cerebral tissue. Frozen sections revealed chronic granulomatous inflammation. Some of the tissue was submitted to the Bacteriology Department for cultures.

Permanent histological sections disclosed large areas of dense fibrous and collagenous connective tissue infiltrated with collections of lymphocytes, mononuclear cells, scattered eosinophiles, and multinucleated giant cells (Fig. 1C). Portions of adjacent brain tissue manifested an intense reactive gliosis. Ziehl-Neelsen, Gram and Giemsa stains failed to reveal specific organisms. The first attempt to stain organisms by the periodic acid-Schiff method failed. On second attempt, however, organisms were found which consisted of a septate fungus showing occasional spores (Fig. 1D). These organisms were found within giant cells and deep within the tumor tissue and could not possibly be considered to be a contaminant.

Unfortunately, cultures were negative for fungi, but the organisms were considered to be morphologically compatible with *Aspergillus*.

A late review of microscopic specimens of the nasal polyps did not reveal a lesion as described above.

(2) *Necropsy* (P.M. 9397). At autopsy part of the right frontal lobe had been surgically removed. A large, firm, irregular, gray tumor mass was firmly attached to the orbital gyri of both frontal lobes anteriorly and to the lesser wing of the sphenoid bone posteriorly. This mass was larger on the right and measured 8×6×3 cm. Part of the tumor was torn away during removal because of the firm attachment to bone. Granulomatous tumor tissue obscured the optic chiasm and involved cranial nerves I, II, III, IV and ophthalmic division of V on the right side (Fig. 2).

Horizontal sections of the brain revealed invasion and destruction of frontal cortex and granulomatous inflammation extending 2 cm. into the frontal centrum. In addition there was evidence of recent bilateral softening of the centrum ovale. This was apparently the result of vascular obstruction by the granuloma. Sections of the tumor mass revealed a dull yellow surface with areas of gray and pink, tough, rubbery tissue. A thick, gray exudate covered the basilar pons to the region of the bifurcation of the vertebral arteries, but there was none elsewhere.

Microscopically the same type of granulomatous inflammation was found as described for the surgical specimens. Periodic acid-Schiff stains revealed the same septate fungus within foreign body giant cells in most areas. In addition, there was microscopic evidence of a chronic, granulomatous meningitis of the base of the pons. The centrum ovale of both frontal lobes showed recent softening, as was evident on gross examination.

Although 8 hours had elapsed between death and autopsy, and the brain had inadvertently been placed in formalin, a section was taken deep within the tumor mass for a second

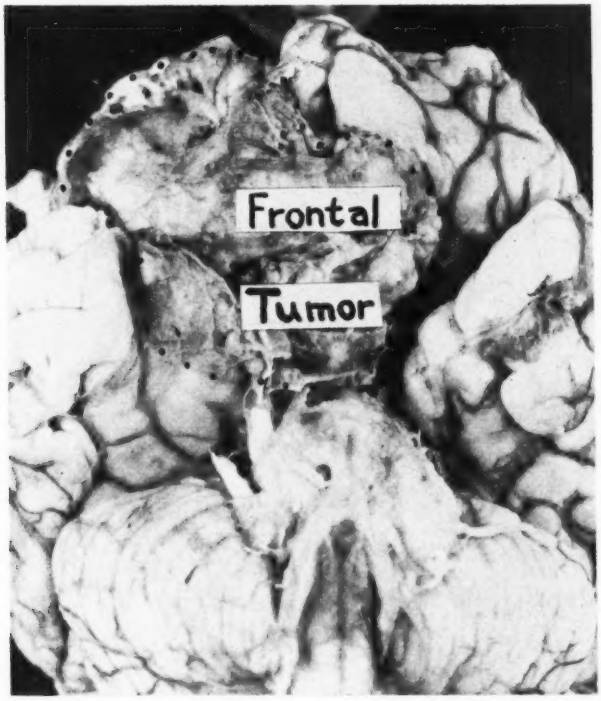


FIG. 2. *Case 1.* Autopsy specimen (9397) showing the large *Aspergillus* granuloma attached to the inferior frontal region of the brain. The tumor is outlined by dotted line.

attempt to culture this fungus. However, this effort also proved fruitless. It was considered that failure to culture the organism before and after death could have been attributable to the very dense type of tissue within which the organisms were found.

The tumor was considered to be a chronic granuloma caused by a septate fungus morphologically compatible with *Aspergillus.*

Case 2. JSH #38283. A white female was first admitted to John Sealy Hospital at the age of 10 on June 8, 1932 under the care of the late Dr. A. O. Singleton, Sr. She had a 2 months' progressive history of a left painless nonpulsating exophthalmus. A decompression of the orbital cavity was performed on June 12, 1932. A very hard tumor was found lying on the floor of the orbital cavity, adherent to the periosteum extending to the medial wall. It was felt that this growth arose from the periosteum and not the nerve.

Pathological Report (SP 5573). The tissue taken from biopsy was described as follows: "The tumor represents an inflammatory process in the orbital cavity. There are numerous giant cells in the sections and a fair number of eosinophiles. There is old inflammatory tissue along with new connective tissue which probably represents the periosteum of the orbital cavity." The diagnosis of nonspecific inflammatory tissue was made.

2nd Admission. The patient was readmitted on July 19, 1932 for further studies. The peripheral blood, serology, sputum, basal metabolic rate, and EKG were normal.

Pathological Report. The diagnosis of the tissue described above (SP 5573) was changed, for a reason unknown to us, to benign giant cell tumor of the orbit.

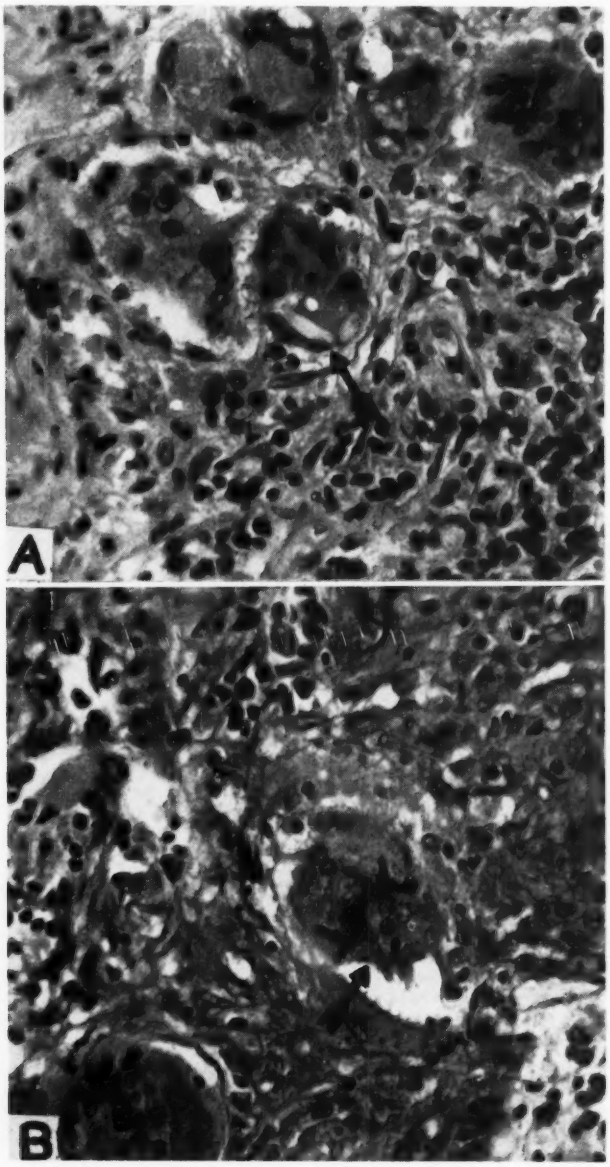


FIG. 3. *Case 2.* (A) Hematoxylin and phloxine stain of granuloma. Note clear spaces in foreign body giant cells. These spaces contain unstained fungi. ×531. (B) Periodic acid-Schiff stain of another section from the same block of tissue, showing a septate fungus morphologically compatible with *Aspergillus*. ×531.

3rd Admission. She was readmitted on March 27, 1933 because of progression of the exophthalmus. On the following day a Krönlein operation with enucleation of the eye and removal of the growth was done. The tumor had penetrated the lateral wall of the orbit, invading the ethmoid cells. Twenty-five mg. of radium was implanted for 8 hours.

Pathological Report (SP 5936). There were many giant cells of the benign giant cell tumor type. There were areas of marked fibrosis and some increase in the number of blood vessels. The diagnosis of a giant cell tumor was again made.

Course. The patient was seen again in 1935 and 1936 with no appreciable change in her condition.

4th Admission. Twelve years after the 1st admission she was readmitted on May 8, 1944 at the age of 22. About a year before she began to complain of severe left temporal headaches associated with vomiting and fever of 104° to 105°F. Shortly preceding this admission she became aware of weakness of her right hand and some difficulty in speech.

Examination revealed a suggestion of a right facial palsy, aphasia, right upper extremity incoordination, right hyperactive deep tendon reflexes, right positive Babinski sign, and papilledema of the right fundus. While in the hospital, her headaches became more severe, with development of lethargy, mental confusion, right hemiplegia, and bilateral choked disc.

On May 25, 1944, Dr. S. R. Snodgrass carried out a left frontoparietotemporal osteoplastic craniotomy. The dura mater was under extreme pressure. About 1 to 2 cm. below the surface of the cortex a huge solid tumor was encountered. An incomplete removal was carried out, leaving a cavity about the size of a medicine glass. Tumor was still present on the floor of the anterior fossa, medially and posteriorly.

Pathological diagnosis (S 14114) was malignant giant cell tumor with cerebral metastasis.

Course. She improved considerably after this operation and was given 11 deep X-ray treatments. Her improvement continued to where she was able to walk and talk. During the following year she received several small courses of deep X-ray therapy because of recurrent headache and a bulging operative defect. With each treatment she had some improvement but of a less degree each time. She finally succumbed Nov. 9, 1945 of increased intracranial pressure, 18 months postoperatively. As the patient died at home in another city no postmortem examination was obtained.

Microscopic Findings. When the slides in this case were reviewed in 1954 by one of us (K.E.) in the process of re-evaluating all cerebral neoplasms at this institution it was noted that there was considerable inflammatory and dense collagenous tissue forming a stroma between large foreign body types of giant cells. In some areas this did have the appearance of a giant cell tumor, but there was an immense inflammatory reaction and an unusual amount of collagenous tissue. Clear, elongated, oval spaces were seen within some of the foreign body giant cells. These clear spaces showed no evidence of staining with hematoxylin and phloxine (Fig. 3A). The sections were stained by the periodic acid-Schiff reaction. This stain revealed a septate fungus within many of the giant cells and in the stroma (Fig. 3B). In some areas the fungus seemed to be forming small bulbous endings. This fungus had the morphological appearance of *Aspergillus.* Therefore a 12-year-old pathological diagnosis was changed from a giant cell tumor to *Aspergillus* granuloma.

DISCUSSION

Within the past few years there has been an increase in the literature on fungous infections of the central nervous system. This relationship to the current administration of antibiotics has been noted by Wybel.[17] He felt that in his patient the administration of penicillin enhanced the growth of the fungus and cited some references in the literature to support this claim. However, antibiotics played no role whatsoever in our 2 patients. Anamnesis of both patients obviously occurred in pre-antibiotic days. It would appear more likely that the paucity of previous reports is ascribable to the fact that the pathological diagnosis had been misinterpreted and special stains and cultures were not employed.

There would seem to be little question that this is a very slow-growing lesion as evidenced by the anamnesis, surgical lesion and the microscopic picture. With such strong features of chronicity one would anticipate more evidence of calcium deposits either in the roentgenograms or the pathological specimens. This is apparently not so since there has been only one such reported case. The most frequent site of *Aspergillus* as a solitary intracranial mass is the frontal lobe, as occurred in our 2 patients. The clinical features seem to fall into two groups. One is that of a slow-growing tumor and the other a chronic meningitis becoming acute. All have met with fatality. Death has been caused by either acute increased intracranial pressure or acute sepsis of the central nervous system.

It is possible that in our first patient the ocular injury 15 years before admission might well have been the portal of entry. However, the history and objective evidence of "sinusitis" should be taken into consideration as the possible portal of entry. Even though the microscopic studies of the nasal polyps were noncontributory, special studies of the pus were not made and this well could have been an *Aspergillus* sinusitis. These possible portals of entry together with our second patient's orbital granuloma certainly coincide with similar cases in the literature, adding further evidence that this fungus gains access to the brain by means of direct extension. The frequent involvement of the frontal lobe favors the portal of entry through the orbit or nasal passageway.

Our second patient offers considerable speculation regarding granulomas. Fungus granulomas involving the cranium and brain may be mistaken for unusual giant cell tumors of bone. The pathologists who had previously studied these microscopic sections were impressed with the degree of inflammatory reaction. It is interesting to note that the original pathological report in 1932 called this a nonspecific inflammatory tissue, but for some reason not known to the authors this was changed to a benign giant cell tumor. One might suppose that the change was made because they readmitted the patient for superficial laboratory studies and since the findings were normal the diagnosis of inflammation was not compatible. The final diagnosis in 1944 was a giant cell tumor of the bone arising in the orbit and invading the brain secondarily. Organisms had not been previously observed and no cultures were made.

The fact that some fungi do not stain with the ordinary stains in some laboratories makes it easy to see how this could well have been mistaken for an unusual giant cell tumor with secondary inflammation. However, the cells of the stroma are definitely inflammatory and not of the neoplastic type seen in giant cell tumors. The fungi stain particularly well with the periodic acid-Schiff stain. This of course brings up the question as to whether these granulomas are as rare as the literature seems to indicate and whether other previously diagnosed giant cell tumors are not actually granulomas. The fungus stain should be done on all atypical giant cell tumors, especially those that show considerable inflammatory reaction. Whenever the stromal cells of a tumor of this sort are definitely inflammatory and not actual neoplastic cells, one should be suspicious of a granuloma and attempts should be made to find the etiological agent by culture and special stains. Any tissue must be ground to a smooth, preferably pipettable suspension, before reliable bacteriological work can be undertaken. Failure to do this in our first case probably accounts for our negative culture.

SUMMARY

Two patients are presented with large single granulomatous tumor masses in the frontal lobes. The histological examination of the surgical and necropsy material revealed a septate fungus morphologically compatible with *Aspergillus*. Special stains are needed to confirm the diagnosis. The possibility of these granulomas being misdiagnosed as giant cell tumors without the special stains is brought out. The portal of entry in one case was probably either the nasal sinuses or orbit and the other the orbit. The clinical history is slow and progressive. The present day therapy, being surgical, is only palliative.

REFERENCES

1. Baker, R. D. Tissue changes in fungous disease. *Arch. Path.*, 1947, *44:* 459–466.
2. Cawley, E. P. Aspergillosis and the aspergilli. Report of a unique case of the disease. *Arch. intern. Med.*, 1947, *80:* 423–434.
3. Coe, G. C. Primary bronchopulmonary aspergillosis, an occupational disease. *Ann. intern. Med.*, 1945, *23:* 423–425.
4. Conant, N. F., Martin, D. S., Smith, D. T., Baker, R. D., and Callaway, J. L. Manual of clinical mycology. *Philadelphia: W. B. Saunders Co.*, 1944, xiii, 348 pp. (see p. 191).
5. David, M., Charlin, A., Morice, J., and Naudascher, J. Infiltration mycosique à *Aspergillus amstelodami* du lobe temporal simulant un abcès encapsulé. Ablation en masse. Guérison operatoire. *Rev. neurol.*, 1951, *85:* 121–124.
6. Grekin, R. H., Cawley, E. P., and Zheutlin, B. Generalized aspergillosis. Report of a case. *Arch. Path.*, 1950, *49:* 387–392.
7. Guillain, G., Bertrand, I., and Lereboullet, J. Étude anatomo-clinique sur un abcès mycosique du lobe frontal. *Rev. neurol.*, 1935, *64:* 684–689.
8. Iyer, S., Dodge, P. R., and Adams, R. D. Two cases of aspergillus infection of the central nervous system. *J. Neurol. Neurosurg. Psychiat.*, 1952, n.s. *15:* 152–163.
9. Just, E. Aspergillusabszess des Grosshirns. *Mitt. Grenzgeb. Med. Chir.*, 1931, *42:* 540–549.
10. McKee, E. E. Mycotic infection of brain with arteritis and subarachnoid hemorrhage. Report of case. *Amer. J. clin. Path.*, 1950, *20:* 381–384.
11. Moniz, E., and Loff, R. Aspergillose cérébrale. *Pr. méd.*, 1931, *39:* 273–274.
12. Oppe. Zur Kenntniss der Schimmelmykosen beim Menschen. *Zbl. allg. Path. path. Anat.*, 1897, *8:* 301–306. (Cited by Iyer, *et al.*[8])
13. Peet, M. M. Aspergillus fumigatus infection of the cerebellum. *Trans. Amer. neurol. Ass.*, 1946, 165.
14. Rénon, L. Étude sur l'aspergillose chez les animaux et chez l'homme. *Paris: Masson & Cie.*, 1897, xii, 300 pp.
15. Thom, C., and Raper, K. B. A manual of the aspergilli. *Baltimore: Williams & Wilkins Co.*, 1945, ix, 373 pp.
16. Wätjen, J. Zur Kenntnis der Gewebsreaktionen bei Schimmelmykosen. *Virchows Arch.*, 1928, *268:* 665–685. (Cited by Iyer, *et al.*[8])
17. Wybel, R. E. Mycosis of cervical spinal cord following intrathecal penicillin therapy. Report of a case simulating cord tumor. *Arch. Path.*, 1952, *53:* 167–173.

A CASE OF CAROTID-BASILAR ANASTOMOSIS

WITH MULTIPLE ASSOCIATED CEREBROVASCULAR ANOMALIES

J. P. SCHAERER, M.D.

Neurosurgical Service, Memorial Hospital, Syracuse, New York

(Received for publication September 16, 1954)

The first report of a case of carotid-basilar anastomosis, as far as the writer knows, was by Quain[10] in 1844. From then until 1950, 14 more cases observed at autopsy were reported in the literature. The advent of angiography made it possible to diagnose this condition during life, and this possibility was mentioned by Hasenjäger[4] in 1937. However, not until 1950 was there a report of a case in which the diagnosis was actually made by this method (Sutton[13]). Another case is known to have been demonstrated angiographically by Sugar[11] (not published). Harrison and Luttrell[3] published 3 more angiographically demonstrated cases in 1953, in 1 of which postmortem examination was also made.

In 4 of the 20 cases so far described in the literature, the diagnosis was made during the patient's lifetime by means of angiography. One more case is known to have been angiographically observed by Sugar. Postmortem examination was made in 18 cases. Of the angiographically diagnosed cases, autopsy was performed in 2.

Sutton[13] found his case among nearly 1000 angiograms. Harrison and Luttrell's[3] 3 cases were encountered in a series of 582 cerebral angiograms. Lindgren[5] found none in 1526 cases. The case reported here was the only one in a series of nearly 600 angiograms.

CASE REPORT

E.B., a 40-year-old white male, was admitted to Syracuse Memorial Hospital with the chief complaint of convulsions, headache and diplopia.

Until 2 days prior to admission, he had been in his usual state of good health when, at 3:00 a.m., he was awakened by a severe frontal headache. At 6:00 a.m. his wife noted he was breathing heavily and foaming at the mouth, with eyes rolled upward. He had urinary and fecal incontinence. When he regained consciousness, he felt weak, but carried out his usual Sunday activities with severe distress. The headache became increasingly severe and empirin gave no relief. The following day he noticed a sudden onset of diplopia in the late afternoon. This was accompanied by an increase in the severity of the headache.

Examination. On admission the patient was alert, cooperative, and well-oriented. Except for a 6th nerve paralysis on the left, the cranial nerves were normal. There was moderate nuchal rigidity. Babinski's reflex was positive bilaterally.

Spinal tap yielded bloody spinal fluid (156000 RBC).

EEG revealed a moderate bilateral slight dysrhythmia with some localization toward the right.

Angiography (Fig. 1) demonstrated a berry aneurysm originating from the left middle cerebral artery about 2 cm. above the bifurcation of the internal carotid artery. In addition, a persistent carotid-basilar anastomosis was clearly demonstrated and the posterior cerebral and basilar arteries were visualized by carotid injection.

Course. On the following day, the patient became suddenly comatose; his respirations deepened, and became irregular and stertorous. He expired within 30 minutes, at 2:30 a.m.

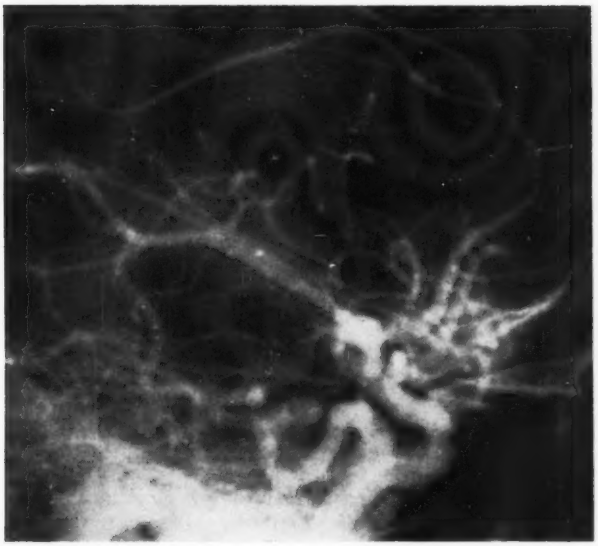


FIG. 1. Angiogram, lateral view.

FIG. 2. Ventral view of brain stem showing small vertebral arteries and basilar artery below anastomosis.

Autopsy was limited to the head. It disclosed the following cerebrovascular anomalies:

1. Persistent carotid-basilar anastomosis on the right, arising from the internal carotid artery in its intradural portion. It was seen to swing medial to the right posterior clinoid process, passing in the midline down to emerge in the midline from the dura mater covering the clivus, joining the basilar artery in its mid portion.

2. Both vertebral arteries as well as the basilar artery inferiorly to this junction were found to be very small, the left vertebral artery being even smaller than the right (Fig. 2).

3. Absent posterior communicating artery on the left; the right posterior communicating artery was rather small.

4. There were two anterior communicating arteries joined in the midline by an additional short branch, thus producing an "H"-shaped connection between the two anterior cerebral arteries.

5. Berry aneurysm, measuring 0.5 cm. in diameter, originating from the middle cerebral artery about 2 cm. above the internal carotid bifurcation.

There was a moderate amount of sub-

arachnoid blood clots about the base of the brain. Tentorial herniation was present bilaterally most marked on the left side. The cerebellar tonsils were slightly herniated through the foramen magnum.

DISCUSSION

The case presented certainly bears out the point made by Padget[7] that among individuals with cerebral aneurysm the circle of Willis often shows a primitive state of development. Dandy[2] drew from this the conclusion that congenital aneurysms arise from weakened points representing the site of incompletely involuted embryonic vessels. Our case seems to support this opinion.

Sunderland,[12] Altmann,[1] and Oertel[6] observed a reduction in size of one or both vertebral arteries in the presence of a persistent carotid-basilar anastomosis. In our case likewise, the vertebral arteries were small, the left being particularly thready.

As Padget[7,8] pointed out, the trigeminal artery seems to be only briefly present in the embryo, disappearing by 5 mm. when the posterior communicating artery has formed and taken over the former's role of supply to the cranial end of the basilar artery. Apparently there is no direct relation between the side on which the posterior communicating is absent and the side on which the carotid-basilar anastomosis has persisted. In our case, the posterior communicating artery was absent on the opposite side; in Harrison and Luttrell's [3] case, the posterior communicating arteries were very small in caliber, but patent. The important factor seems to be the total circulatory capacity of the posterior communicating arteries.

In relation to the dorsum sellae, two types of courses of the carotid-basilar anastomosis have been described. In 7 cases, the anastomosis ran through the dorsum sellae; in 9 cases, it skirted the lateral margin of the sella. In our own case, the anastomosis emerged medial to the posterior clinoid process from the dorsum sellae. Padget[9] explains the differences in the exact position and relation of the persistent adult vessel by variable factors in the formation of the dura mater around the sella at the 40 mm. stage embryo. At this point, the dura mater will come in contact with different parts of the vessel in different cases. Often the contact occurs originally relatively near the basilar end of the vessel, and therefore, posterior to the site of the future sella. In these cases, the carotid-basilar anastomosis will make its course through the dorsum sellae. If the contact occurs posterolateral to the future sella, the anastomosis will skirt the lateral margin of the dorsum sellae.

SUMMARY

A case of carotid-basilar anastomosis is presented which was diagnosed arteriographically during life and which was examined post mortem. Concurrent multiple cerebrovascular anomalies are described and discussed.

REFERENCES

1. Altmann, F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance. Report of a new case of persistent stapedial artery in man. *Laryngoscope, St. Louis*, 1947, *57:* 313–339.
2. Dandy, W. E. Intracranial arterial aneurysms. *Ithaca, N. Y.: Comstock Publishing Co., Inc.*, 1944, viii, 147 pp.
3. Harrison, C. R., and Luttrell, C. Persistent carotid-basilar anastomosis. Three arteriographically demonstrated cases with one anatomical specimen. *J. Neurosurg.*, 1953, *10:* 205–215.
4. Hasenjäger, T. Ein Beitrag zu den Abnormitäten des Circulus arteriosus Willisii. *Zbl. Neurochir.*, 1937, *2:* 34–39.

5. LINDGREN, E. Percutaneous angiography of the vertebral artery. *Acta radiol., Stockh.*, 1950, *33:* 389–404.
6. OERTEL. Über die Persistenz embryonaler Verbindungen zwischen der A. carotis interna und der A. vertebralis cerebralis. *Anat. Anz.*, 1922, *55* (Verhandl. anat. Gesellsch.): 281–295.
7. PADGET, D. H. The circle of Willis. In: Dandy[2] (see pp. 67–90).
8. PADGET, D. H. The development of the cranial arteries in the human embryo. *Contr. Embryol. Carneg. Instn. 212*, 1948, *32:* 205–261.
9. PADGET, D. H. Personal communication.
10. QUAIN, R. The anatomy of the arteries of the human body and its applications to pathology and operative surgery, with a series of lithographic drawings. *London: Taylor & Walton*, 1844, xv, 550 pp. (see p. 507).
11. SUGAR, O. Cited by Harrison and Luttrell.[3]
12. SUNDERLAND, S. Neurovascular relations and anomalies at the base of the brain. *J. Neurol. Neurosurg. Psychiat.*, 1948, n.s. *11:* 243–257.
13. SUTTON, D. Anomalous carotid-basilar anastomosis. *Brit. J. Radiol.*, 1950, n.s. *23:* 617–619.

ARACHNOIDAL CYSTS OF THE CEREBELLOPONTINE ANGLE

FRANCISCO GARCÍA BENGOCHEA, M.D.,* AND FRANCISCO LEÓN BLANCO, M.D.
Havana, Cuba

(Received for publication September 17, 1954)

Arachnoidal cysts of the cerebellopontine angle attaining sufficient size to cause the symptomatology of a tumor are rather rare. In 1953 Nichols and Manganiello[11] reported a case of traumatic arachnoidal cyst simulating an acoustic neurinoma, and they thought that this represented a case in which the cyst was in a location not described previously.

We know of no other report dealing with this type of lesion in such location except that of Aubry and Ombrédanne[2] in their monograph *Étude Oto-Neurologique et Chirurgicale du Vertige.* They classified cerebellopontine arachnoiditis in three groups: the pseudotumoral, the cystic, and the adhesive form. The pseudotumoral form is characterized by a large cyst giving a full-blown picture of a cerebellopontine angle tumor, most commonly an acoustic neurinoma. As an example of this type or form they referred to a case of Monier-Vinard, Ramadier, and Chaussé, but they did not mention any personal observation. The cystic form, which consists of a small cyst in the region of the internal auditory meatus, presents a clinical picture limited to cochleovestibular manifestations associated with some mild neurological disturbances such as diminution of the corneal reflex on the same side. They reported 5 personal observations. Obviously, the difference between the pseudotumoral and the cystic form of Aubry and Ombrédanne is a matter of degree in the size of the cyst and the compression that it exerts on neighboring neural structures.

The case reported by Thompson[12] presented primarily a lesion lying posteriorly or dorsally to the right cerebellar hemisphere and crossing over the vermis somewhat to the left of the midline.

In speaking of the differential diagnosis of acoustic nerve tumors, Craig[7] referred to the localized cystic arachnoiditis of the cerebellopontine angle which "may mimic the signs and symptoms of tumor so completely that differentiation is impossible." He gave no specific reference and did not refer to any personal observation.

In the case described by Alexander[1] there was a midline lesion in the subtentorial supracollicular region and the main clinical findings were related to the marked hydrocephalus in a child 18 months old.

Other reports such as those of Horrax,[9] Kaplan,[10] Demel,[8] Bucy,[4,5] and Trowbridge and French[13] do not deal specifically with arachnoidal cysts of the cerebellopontine angle.

Our patient presented a full-blown cerebellopontine angle syndrome with marked evidence of increased intracranial pressure. The cyst occupied the angle, displaced the left cerebellar hemisphere medially, and reached the pons, extending rostrally to the tentorium and caudally to a level midway between the jugular foramen and the foramen magnum. We cannot establish a definite etiological factor although there was a history of a purulent process of the left ear which, from the data available, seemed to correspond to an external ear infection.

* Calle 5ª No. 462, Vedado, Habana, Cuba.

In the other reported cases of arachnoidal cysts of the posterior fossa, local or distant inflammatory processes, trauma, subarachnoidal bleeding, and congenital malformations of the hindbrain have been mentioned as possible etiological factors in the mechanism of production of this type of cyst.

CASE REPORT

P.N.T., a 51-year-old white married female, was seen for the first time on Oct. 4, 1952, with a history of tinnitus and loss of hearing in the left ear. These symptoms began sometime between 1929 and 1933. The patient was not certain of the exact date of their onset and she could not tell which appeared first, yet she was positive that in 1934 she had consulted an otorhinolaryngologist, Dr. Tapia, in Spain, concerning these symptoms. They persisted since then and apparently became worse in the latter part of 1951.

In 1936 she felt for the first time a paroxysmal shooting pain in the distribution of the 2nd and 3rd divisions of the 5th cranial nerve. That year she had several episodes of such pain, with recurring spells in 1947 and in March, 1952. Her description of the pain corresponded to that of tic douloureux of the 2nd and 3rd divisions of the left 5th cranial nerve.

In the latter part of 1950, three new symptoms set in. One was a true vertigo, at first occasional but which became progressively worse particularly after May, 1952, when it occurred several times during the day, especially when she bent forward. Simultaneously with the vertigo she began to suffer from instability of gait with a tendency to fall to the left. This symptom became much more marked after May, 1952, and it made her walking extremely difficult. With the onset of her vertigo and gait difficulties and following a parallel course with them, she started to notice a pounding sensation inside her head, especially towards both frontal regions. This sensation became more severe and frequent after May, 1952. It was also precipitated or aggravated by bending forward.

In May, 1952, the patient began to complain of a marked sensation of pressure behind both eyes and in the last week of August she noticed for the first time a transitory and partial loss of vision lasting from 4 to 8 minutes and recurring from 5 to 6 times during the day.

In her past personal history she referred to a purulent process of her left ear which began some time in 1920 or 1921. At that time she was seen by two otorhinolaryngologists who prescribed zinc oxide ointment and the process disappeared in the course of a few months. The patient believed that she had an eczema of the outer ear, but since both specialists died more than 12 years ago, we have been unable to obtain accurate data of this process. The patient denied any other ear infection as well as cranial trauma.

Examination revealed the following abnormalities: (1) A markedly unsteady gait with a definite tendency to veer and fall to the left. (2) A consistent fall to the left on the Romberg maneuver. (3) *Décomposition des mouvements* and dysmetria, both in the left upper and lower extremities and to a lesser degree in the right extremities. A moderate intentional tremor was noted in the left upper extremity. (4) Dysdiadokokinesia of the left upper extremity. (5) Spontaneous nystagmus increased in both lateral gazes. (6) Visual acuity of right eye, 20–40; of left eye, 20–25. There was marked bilateral papilledema: 5 D. on right and 4 D. on left. The edges of the left optic disc were infiltrated by blood and there was marked venous ingurgitation with an A-V ratio of 1 to 4. (7) Bilateral diminution of corneal reflex, more marked on the left. (8) Moderate weakness of all the left facial muscles.

Otological examination performed by Dr. Miguel A. Abalo revealed a dead labyrinth on the left side and marked diminution of auditory acuity on the left with practically normal acuity on the right. Plain x-rays of the skull with special views of the petrous bone and internal auditory meatus showed no abnormality.

In view of the history and physical findings a lumbar puncture was considered unnecessary and perhaps even dangerous. The clinical diagnosis of a space-occupying lesion of the left cerebellopontine angle was quite evident and we thought we were dealing with an acoustic neurinoma. It was recommended that the patient be operated on in the next 3 or 4 days; nevertheless, for family reasons, surgery was delayed 2 weeks.

Operation. On Oct. 18, 1952, the patient was scheduled for ventriculography and suboccipital craniotomy at the Centro Médico Quirúrgico de la Habana. Scopolamine (0.0004 gm.) was given subcutaneously 30 minutes before she was taken to the operating room. At that time she was fully conscious and responded readily to commands. Morphine was not given intentionally. On arrival at the operating room she was unresponsive to commands and would react very little to pain stimulation. Her pulse rate was markedly diminished. She was rapidly intubated and placed face-down in the ordinary suboccipital or cerebellar position. We usually prefer to perform operations for angle tumors with the patient in the side or in the upright position, but the emergency created by her unresponsiveness rushed us to place her in the face-down position for occipitoparietal burr holes and puncture of the lateral ventricles.

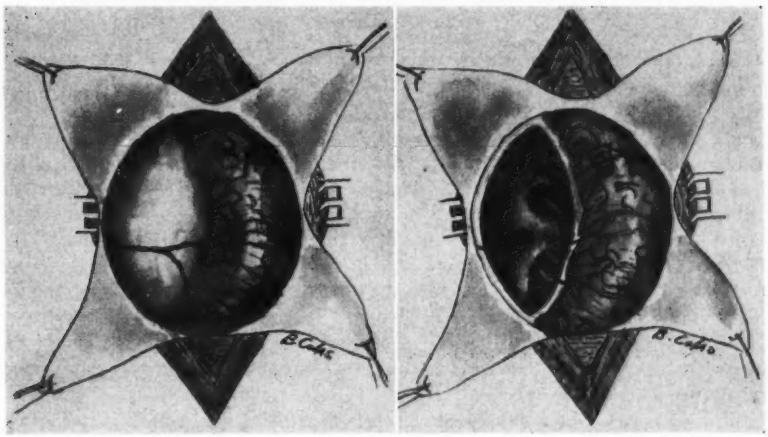
The left lateral ventricle was entered with a brain cannula at 4½ cm. from the skin surface. The fluid spurt under great pressure. Another burr hole was placed in the right occipitoparietal region and a rubber catheter was inserted into the right lateral ventricle which was also dilated. The brain cannula was withdrawn from the left lateral ventricle but the rubber catheter in the right lateral ventricle was fixed and left in place all during the operation and it was opened intermittently for further reduction of pressure.

A left hemisuboccipital craniectomy was performed through a straight lateral and vertical incision, as advocated by Bucy. The dura mater was under great pressure which was reduced by opening the rubber catheter in the right lateral ventricle. The bone removal extended laterally to the mastoid cells, medially until close to the midline, and caudally to about 1 cm. from the foramen magnum, the rim of which was not resected.

When the dura mater was opened, the left cerebellar hemisphere was found displaced toward the midline by a whitish, smooth, rounded mass with the appearance of a cyst (Fig. 1). At first glance, we thought it was one of those cysts that sometimes cover part of the acoustic neurinomas, but when we opened its presenting wall, a clear, colorless, and transparent fluid flowed under pressure. As we aspirated this fluid, we found to our surprise a huge cystic cavity extending laterally to the mastoid and petrous bone, medially to the ventrolateral aspect of the pons, rostrally to the edge of the tentorium, and caudally to about 2 or 3 mm. from the exit of the left 12th cranial nerve. The cavity of the cyst measured 4 cm. in width, 3½ cm. in height, and 5 cm. in depth. Its wall was formed all around by a paper-thin, whitish and slightly transparent membrane which had the gross appearance of arachnoidal structure. It covered the 5th, 7th, 8th, 9th, 10th, and 11th cranial nerves which appeared flattened, especially the 7th and 8th. The lateral surface of the left cerebellar hemisphere was displaced medially to a distance almost midway between the inner table of the mastoid and the midline. A concavity was formed on the lateral surface of the left cerebellar hemisphere, replacing its normal convexity. No evidence of tumor was found and about ⅗ of the wall of the cyst was removed with the aid of scissors (Fig. 2). It was practically an avascular structure with the exception of a small vessel which crossed its posterior surface, running between the cerebellar hemisphere and the angle. The portions of the membrane adhering to the cranial nerves and to the pons were not resected in order to avoid trauma to these structures. Patency of the ventricular system was tested by allowing fluid to flow by gravity through the rubber catheter into the right lateral ventricle and watching for its free flow into the cisterna magna, the left lateral portion of which was exposed. At first, very little retraction was needed on the cerebellum, which began to expand before closure was started. The dura mater was closed with interrupted silk sutures and the rest of the wound was closed in layers in the usual manner. The catheter was withdrawn from the right lateral ventricle before applying the dressing.

Microscopic Examination. The cyst wall was formed by a thin membrane of collagenous fibers and both its inner and outer surfaces were lined by cells with the histological appearance and arrangement of arachnoidal cells (Fig. 3).

Postoperative course was uneventful. The patient was conscious that same night, was allowed to get out of bed on the 4th postoperative day, and, on discharge from the hospital a week after operation, she was able to walk without help.



FIGS. 1 and 2. (*Left*) Artist's drawing of the appearance of the lesion after the dura mater was opened. (*Right*) Part of the dorsal wall of the cyst has been removed allowing visualization of its cavity. The 7th, 8th, 9th, 10th, and 11th cranial nerves are covered by the ventrolateral wall of the lesion.

The symptoms and signs continued to recede and by December 2, 1952 the neurological findings were within normal limits with the exception of a mild unsteadiness in gait, diminished hearing and a dead labyrinth in the left ear, and occasional mild tinnitus. The papilledema disappeared completely leaving no secondary optic atrophy.

The patient was not seen from March 21, 1953 until June 10, 1954, when she was asked to come to the office for a check-up visit. The residual symptoms just mentioned have persisted. Ever since the operation she has been able to carry on a happy and active life.

FIG. 3. Photomicrograph of section of the cyst wall. Hematoxylin-eosin stain, ×190.

DISCUSSION

In the English medical literature we have found only one report of an arachnoidal cyst of the cerebellopontine angle simulating a tumor of such location and that is the case reported by Nichols and Manganiello.[11]

The French authors Aubry and Ombrédanne[2] entered into a more detailed description of cerebellopontine arachnoiditis and they described 5 personal observations of their cystic form verified at operation. In those 5 cases the symptomatology was almost always restricted to the 8th cranial nerve. Of their pseudotumoral form they gave no personal experience, but they referred to a case of Monier-Vinard, Ramadier, and Chaussé.

In the rest of the medical literature that we have reviewed, we have not found any other communication about this specific type of lesion. Therefore our report may be of interest in the sense that it represents the third reported case, as far as we are aware, in which the cyst was in the cerebellopontine angle and gave the full-blown picture of a tumor.

As we said before, the cystic form of cerebellopontine arachnoiditis described by Aubry and Ombrédanne[2] seems to be essentially the same lesion as the pseudotumoral form, the only difference being the size of the cyst, which is small in the cystic form and large in the pseudotumoral form. The French otologists apparently are quite aware of the cystic form, since there are other monographs, such as that by Bourgeois,[3] in which reference is made to this type of pathology.

We cannot establish a definite etiological factor in our case and can only point out that the patient had a purulent process of the left external auditory meatus which occurred about 8 to 10 years before the onset of the first symptoms, tinnitus and loss of hearing on the left. If this purulent process had occurred in the middle ear we should be less hesitant to consider it as the source of the inflammatory process accounting for the arachnoidal reaction and the formation of the cyst, but it is more difficult, although theoretically possible, to consider a purulent process of the external ear the source of an arachnoiditis.

SUMMARY

The subject of cystic arachnoiditis of the cerebellopontine angle is reviewed.

A case of a verified arachnoidal cyst in the cerebellopontine angle producing the clinical picture of a cerebellopontine angle tumor with obstructive hydrocephalus is reported. So far this seems to be a very rare type of lesion and we know of only 2 similar cases previously recorded in the medical literature. If any etiological factor can be adduced in our case, it would be an arachnoiditis secondary to an inflammatory process of the external auditory meatus.

Surgical removal of part of the wall of the cyst produced excellent improvement which has been maintained up to date, 22 months after the operation.

REFERENCES

1. Alexander, E., Jr. Benign subtentorial supracollicular cyst as a cause of obstructive hydrocephalus. Report of a case. *J. Neurosurg.*, 1953, *10:* 317–323.
2. Aubry, M., and Ombrédanne, M. Étude oto-neurologique et chirurgicale du vertige. Indications et résultats de la chirurgie intracranienne du nerf auditif. *Paris: Masson & Cie*, 1937, 248 pp. (see pp. 100–122).
3. Bourgeois, R. Les hydrocéphalies aiguës et subaiguës d'origine otique. Accidents méningés otogènes purement hypertensifs. *Paris: Masson & Cie*, 1935, 178 pp. (see pp. 28–29, 110–122).

4. Bucy, P. C. Intrinsic tumors of the cerebellum and brain stem. In: *Surgical treatment of the nervous system*. F. W. Bancroft and C. Pilcher, Ed. Philadelphia: J. B. Lippincott Co., 1946, xvii, 534 pp. (see pp. 204–238).
5. Bucy, P. C. Surgical treatment of acoustic tumors. *J. Neurosurg.*, 1951, *8:* 547–555.
6. Craig, W. McK. Chronic cystic arachnoiditis. *Amer. J. Surg.*, 1932, n.s. *17:* 384–388.
7. Craig, W. McK., and Shelden, C. H. Tumors of cranial nerves. In: *Surgical treatment of the nervous system*. F. W. Bancroft and C. Pilcher, Ed. Philadelphia: J. B. Lippincott Co., 1946, xvii, 534 pp. (see pp. 132–153).
8. Demel, R. Die Meningitis serosa circumscripta cerebralis unter dem Bilde des Hirntumors und ein Beitrag zu ihrer Ätiologie. *Arch. klin. Chir.*, 1923, *125:* 561–577.
9. Horrax, G. Generalized cisternal arachnoiditis simulating cerebellar tumor: its surgical treatment and end-results. *Arch. Surg., Chicago*, 1924, *9:* 95–112.
10. Kaplan, A. Pia-arachnoidal cysts of the posterior fossa. *Amer. J. Surg.*, 1948, n.s. *76:* 102–106.
11. Nichols, P., Jr., and Manganiello, L. O. J. Traumatic arachnoidal cyst simulating acoustic neurinoma. *J. Neurosurg.*, 1953, *10:* 538–539.
12. Thompson, R. K. Cystic cerebellar arachnoiditis. *J. Neurosurg.*, 1946, *3:* 461–467.
13. Trowbridge, W. V., and French, J. D. Benign arachnoid cysts of the posterior fossa. *J. Neurosurg.*, 1952, *9:* 398–404.

OSTEOCHONDROMA OF THE LUMBAR SPINE

REPORT OF A CASE

HUSAMEDDIN GOKAY, M.D.,* AND PAUL C. BUCY, M.D.†
Chicago, Illinois

(Received for publication October 1, 1954)

One of the most common benign tumors of bone is the osteochondroma. These tumors are usually found at the ends of the long bones of the extremities, but they may also arise from any part of the skeletal system, bones of the jaw, skull, hand, and foot. The vertebral column is rarely a site for these lesions. Meyerding[5] reported the removal of 265 exostoses from 235 patients and among these only one tumor was located in the lumbar spine. In 310 cases reported by Geschickter and Copeland,[2] the tumors involved the vertebrae in 10 cases. Another interesting case of osteochondroma related to the vertebral column was discussed by Ilgenfritz.[3] In this latter case the osteochondroma grew from the 7th cervical vertebra anteriorly and it was diagnosed as a thyroid tumor before the exploration.

The following unusual case of osteochondroma arising from the lumbar spine exhibited the signs and symptoms of compression of the cauda equina.

CASE REPORT

Mrs. V.H., a 24-year-old white housewife, referred by Dr. Donald B. Douglas of Waukegan, Illinois and Dr. E. A. Kahn of Ann Arbor, Michigan, was first admitted to the Neurosurgical Service at The Chicago Memorial Hospital on Feb. 28, 1950. Her main complaints consisted of a hard mass attached to the lumbar spine, weakness and numbness in the left leg, urinary incontinence and constipation.

She stated that she had been in good health until she was 6 years old when a hard mass began to grow on her lumbar spine; later similar masses appeared about both knees, the left ankle, and the left humerus. These masses increased slowly in size. About 1 year later, part of the mass in the lumbar region was excised for cosmetic reasons. She had had no neurological symptoms pertaining to these masses until 1945 when pain developed in her buttocks and lower extremities. The left leg became numb and weak. She was placed on bed rest with the diagnosis of possible rheumatic arthritis. She was symptom free in about a month. One year later she was hospitalized with pneumonia during which time weakness developed in her left foot and toes, and this weakness continued thereafter. In 1948 she became pregnant. She had a normal delivery but subsequently she complained of constant constipation which required cathartics. In September 1949 she started to have pain in her left lower lumbar region which radiated down to the lateral aspect of the left thigh. The pain in the back was constant for about 1 month. Five months before admission her whole left lower extremity became numb and remained so. Two months before admission she began to have constant dribbling of urine during the night together with urgency and frequency in the daytime. At about the same time some numbness developed in the right foot.

The family history revealed that her father and her brother, and also a grandfather, had bony masses removed from their legs.

* Now at Indiana University Medical Center, Indianapolis, Indiana.

† Northwestern University Medical School, and the Chicago Wesley Memorial Hospital, Chicago, Illinois.

Examination. The patient was well developed, well nourished, alert and cooperative. Her blood pressure was 110/50, pulse 100, and temperature 98.8°. There were bony masses in the left forearm, on the medial aspect of the right knee, above and below the joint, on the lateral aspect of the left thigh, below the left knee joint on the medial side, in addition to a large hard mass in the lower lumbar region. Hypalgesia and hypesthesia were found below the level of the 5th lumbar dermatome on the left side and below the 3rd sacral on the right. There was a slight loss of position and vibratory sensibilities in the left lower extremity. There was

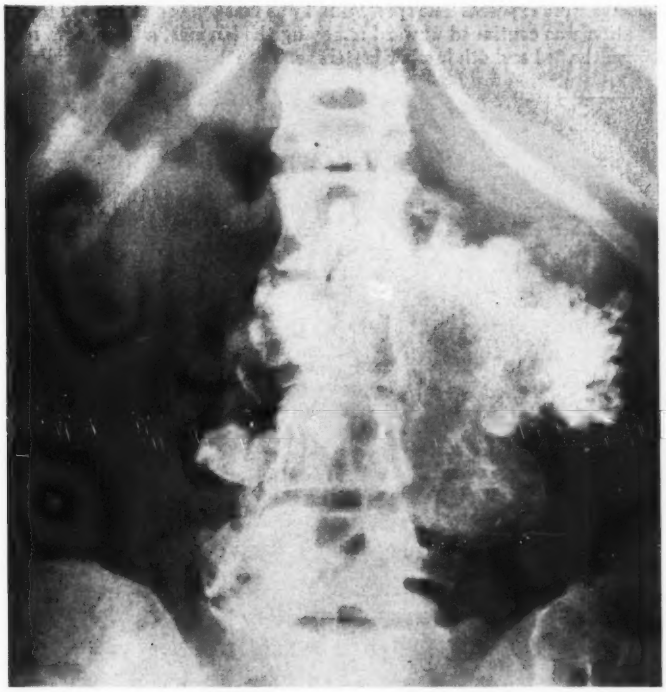


FIG. 1. Preoperative condition showing osteochondroma of the lumbar spine extending much more to the right than to the left.

slight weakness of the biceps and triceps muscles in the left arm. There was definite weakness in the left leg, slight in the quadriceps and hamstrings, but marked in dorsiflexion of the foot and the big toe. There was slight atrophy of the right thigh and calf. The abdominal reflexes were absent. The left knee jerk was decreased and the left ankle jerk was absent. Babinski's sign was not elicited. She walked with a slow, uncertain gait, limping on the left leg.

The blood count and urine were normal. Wassermann and Kahn tests on the blood were negative.

Roentgenograms of the lumbar spine showed a large irregular calcified mass with varying degrees of density and a rather smooth scalloped border which arose from the lumbar spine. The mass was particularly large on the right side, where it extended for a distance over 7.5 cm. lateral to the vertebrae. The process involved the 2nd, 3rd, 4th, and 5th lumbar vertebrae (Fig. 1). Films of both knees revealed multiple exostoses arising both from the distal ends of the femora and the proximal ends of the fibulae. There were multiple exostoses at the

proximal end of the left humerus. An intravenous pyelogram showed a normal left kidney, pelvis, calyx and ureter; on the right side the pelvis and calyx system was at the level of the 3rd and 4th lumbar vertebrae and apparently was pushed downward by a mass.

1st Operation. On March 2, 1950, with the patient under general anesthesia, the lesion in the lumbar region was exposed through a midline skin incision. The mass consisted of cancellous bone and cartilaginous tissue. The mass lying directly over the spine and nodules of tumor lying to the left of the spine were gradually removed piecemeal. A ridge of tumor was left on the right side of the spine to act as a splint. The laminae of the lumbar vertebrae from L2 to L5 inclusive were exposed. The spinal canal was first opened at the 5th lumbar lamina. As the exploration was continued upward removing the laminae, a large, firm mass was uncovered between the 3rd and 4th lumbar vertebrae. The mass extended laterally through the

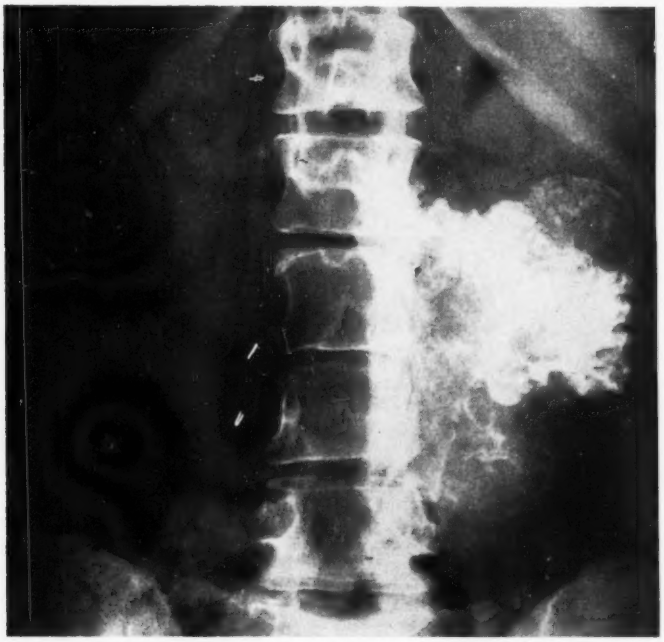


FIG. 2. Condition following the first operation—all tumor to the left of the lumbar spine and in the spinal canal has been removed.

greatly dilated intervertebral foramen and connected with a mass approximately 3 cm. in diameter lying to the left of the spine at this level. This had the appearance of an hourglass neurofibroma but it was actually composed of firm, almost transparent bluish-white cartilage. Lying just above this mass there was a mass of cartilage and bone arising from the laminae of the 3rd lumbar vertebra and protruding downward into the spinal canal. This tissue practically filled the canal at this point. The dural sac was pressed downward and laterally toward the right. A few of the nerve fibers of the cauda equina were flattened against the vertebral bodies, and a few of them had been forced far laterally out of the spinal canal and lay actually against the paravertebral muscles. All of the osteochondroma was carefully removed from the spinal canal so that the cauda equina was completely decompressed. The part of the tumor on the right side of the lumbar spine was left for a 2nd operation.

Course. The patient did well except for urinary retention, but she was able to urinate with the help of urocholine. She was up and walking on the 12th postoperative day. The urinary retention cleared shortly after her discharge from the hospital on March 23, 1950.

2nd Admission. On Oct. 1, 1950 she was re-admitted for the 2nd stage of the operation. She still complained of constipation, occasional urinary incontinence, weakness of the left leg, low back pain and also sharp radiating pains in the left leg. These radiating pains in the leg had developed only 1 month previously.

Examination revealed marked improvement over the findings of the 1st admission. There was bilateral hypalgesia in the 3rd to the 5th sacral dermatomes. There was moderate weak-

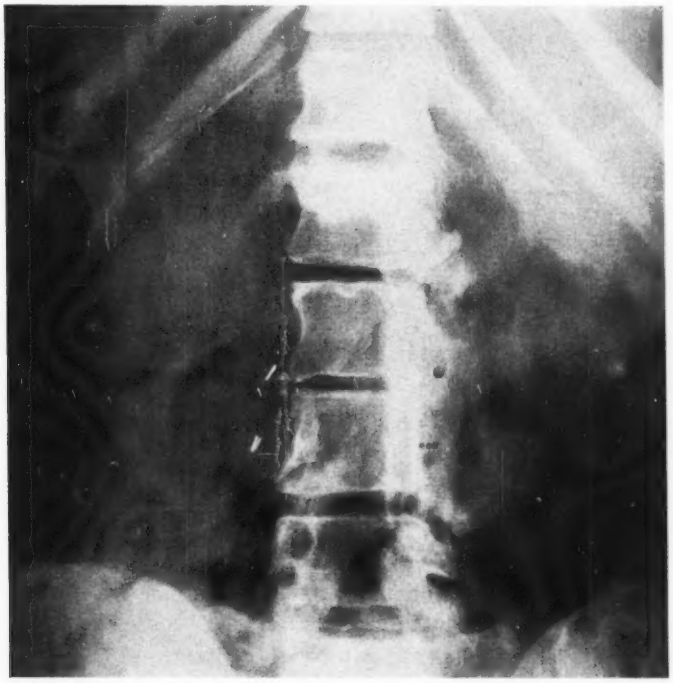


FIG. 3. Condition following the second operation—most of the tumor to the right of the spine has also been removed but a ridge of bone and tumor has been left in the region of the articular facettes to form support for the spine.

ness of all movements of the left leg with some slight atrophy. The left knee jerk was decreased and the ankle jerks were absent on both sides.

Roentgenograms of the lumbar spine showed that the osteochondroma had been removed from the left side of the lower lumbar spine together with the laminae of the 3rd, 4th, and 5th lumbar vertebrae, the left pedicle of the 3rd lumbar vertebra and the articular processes between the 2nd, 3rd and 4th lumbar vertebrae (Fig. 2).

2nd Operation. On Oct. 2, 1950, the old vertical incision was reopened and an additional transverse incision was made in the midlumbar region on the right side. The mass of osteochondroma was totally exposed. It lay beneath the superficial muscles of the back; the muscles were dissected and incised transversely. With the aid of a chisel, the mass of bone

and cartilage was removed except for a ridge of bone attached to the transverse processes and pedicles of the lumbar vertebrae on the right side. This was left in place in order to provide a bony splint for the weakened lumbar spine. The mass of cartilaginous tissue seemed to project laterally from the spine between layers of muscle. It extended far forward but not into the retroperitoneal space, so far as could be seen. None of the roots of the sciatic nerve was seen.

Course. The patient did very well, except again for some urinary retention. She was up on the 12th postoperative day and discharged on the 15th.

Subsequently the patient was seen at regular intervals. On July 17, 1951, she stated that she seldom had dribbling of urine and she had no difficulty with constipation. She complained

FIG. 4. Approximately 4 years after the first operation.
There has been no further growth of the tumor.

of no pain except some aching in the back when she was up too long. Her legs and back were strong and she was doing square-dancing. She also stated that she had normal sensation when the bladder and the rectum were distended and also on sexual intercourse, whereas preoperatively she had no sensation. Examination revealed that the lumbar spine was ankylosed, and the left calf was 1.5 cm. smaller than the right one. There was hypesthesia of the 3rd to 5th sacral dermatomes on the right side and of the 5th lumbar and all the sacral on the left. Knee and ankle jerks were absent on the left. Strength and the station and gait were good.

Films of the lumbar spine revealed that almost the entire remaining portion of the large osteochondroma had been removed (Fig. 3).

On Feb. 8, 1954, the patient was seen for a last re-check for this report. She stated that she was doing very well. She did not drag her left foot when walking. She had no difficulty climbing stairs. She complained of only slight backache when she carried heavy laundry baskets. She still had 3–4 ounces of residual urine, and occasionally urinary infections occurred which were readily controlled by sulfonamides. She had some dribbling of urine during her periods and also when she coughed. Her main complaint was of the masses around her knees; these were growing slowly and had begun to give her some discomfort.

Examination revealed normal station and gait. There was no weakness in the right leg, but all movements at the left ankle showed about 25 per cent weakness, and there was slight weakness of dorsiflexion of the left big toe. There was hypesthesia in the 3rd sacral dermatome on the right and the 1st, 2nd, and 3rd sacral on the left. The right calf measured 36 cm. in circumference, the left 33 cm. Both knee and ankle jerks were absent bilaterally.

Films of the lumbar spine showed some calcification but no apparent growth of the tumor mass (Fig. 4).

Pathological Description (Dr. Otto Saphir). The specimen consisted of numerous segments of gelatinous and cartilaginous tissue with spicules of bone. Sections consist of cartilaginous tissue and fibrous connective tissue. The cartilage is deposited irregularly, stained variably and in some places shows small cystic areas and myxomatous changes. Set in lacunae are round cells with some regular nuclei and deeply acidophilic cytoplasm.

DISCUSSION

This young woman had an osteochondroma arising from the lumbar spine and other parts of the skeleton which was apparently familial. The tumor, as it is observed in the X-rays, involved all the lumbar vertebrae except the first one, and the major part of it grew laterally. However, the important part from the neurological standpoint was the tumor that grew into the spinal canal and compressed the cauda equina. Such a development is unusual. Of the 12 cases cited earlier, in only 1 were there symptoms of neurological involvement. Bradford[1] has reported 2 cases in which small osteochondromata arose from the vertebral bodies and pressed backward against the anterior surface of the spinal cord in the thoracic region. These also differed from the case recorded here in that his cases apparently were not instances of diffuse familial osteochondromata.

Although the cauda equina was markedly injured by the tumor in our case, the patient made a remarkable recovery following the removal of the tumor.

These tumors usually occur in young adults and mostly among males. They arise in areas where cartilage is ordinarily found and develop typically at the sites of attachment of tendons or ligaments. There is often a distinct familial history. Many unsubstantiated theories have been advanced as to their etiology, e.g., rickets, trauma, embryonic cell rests,[6] or persistence of cartilage of epiphyseal plate.[4]

The typical osteochondroma has the appearance of a firm lobulated tumor. It consists of cancellous bone interspersed with marrow, attached to its point of origin by a bony pedicle. A clear zone of cartilage covered by a thin layer of fibrous tissue surrounds the tumor. In its deeper portion the cartilage undergoes calcification and abutting on this zone laminated bone spicules are to be found.

Ordinarily these lesions are benign; operative removal may become necessary because of interference with the function of the surrounding structures. A recurrence took place in only 5 per cent of the cases in Geschickter and Copeland's series.[2] Some tumors may undergo malignant change but this is rather rare. In our case even though the tumor was not completely removed, there was no evidence of any recurrence 4 years after the operation.

REFERENCES

1. Bradford, F. K. Intraspinal tumors (report of twelve cases). *Dis. nerv. Syst.*, 1954, *15:* 55–60.
2. Geschickter, C. F., and Copeland, M. M. Tumors of bone. *Philadelphia: J. B. Lippincott Co.*, 1949, 3rd ed., xviii, 810 pp.
3. Ilgenfritz, H. C. Vertebral osteochondroma. *Amer. Surg.*, 1951, *17:* 917–922.
4. Keith, A. Studies on the anatomical changes which accompany certain growth-disorders of the human body. *J. Anat., Lond.*, 1920, *54:* 101–115.
5. Meyerding, H. W. Exostosis. *Radiology*, 1927, *8:* 282–288.
6. Müller, E. Über hereditäre multiple cartilaginäre Exostosen und Ecchondrosen. *Beitr. path. Anat.*, 1913, *57:* 232–281.

THE OBLIQUE VIEW IN CEREBRAL ANGIOGRAPHY

CURWOOD R. HUNTER, M.D.,* AND FRANK H. MAYFIELD, M.D.*
Cincinnati, Ohio

(Received for publication July 30, 1954)

Kinley and Leighninger[1] in 1952 illustrated by means of an oblique view the origin of an aneurysm not clearly defined in routine projections. The conventional lateral and anteroposterior X-ray views utilized in angiography demonstrate intra-

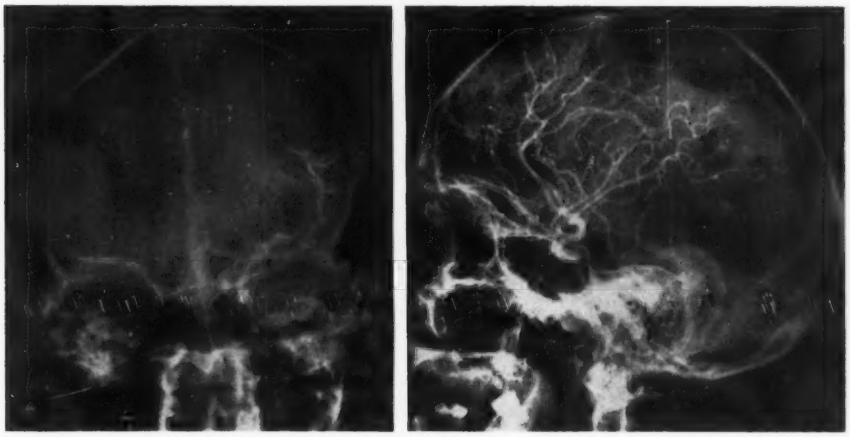


FIG. 1. Anteroposterior and lateral views.

cranial aneurysms and their origins from parent vessels in most instances. A few, however, completely escape detection with these two projections and in some it is difficult, if not impossible, to be sure of the precise origin of the aneurysm in order to properly plan surgical attack. In our experience, the oblique view has been found particularly helpful in arriving at an accurate diagnosis and now constitutes one view of the conventional series. Indeed, in cases in which only limited opportunity to obtain studies is present, such as in the aged or poor risk, this view may be the only one attempted.

Anteroposterior and lateral views in Fig. 1 indicate an apparently normal angiogram. In Fig. 2 the oblique view demonstrates a small aneurysm arising from the middle cerebral artery. The oblique view (Fig. 3), with the head rotated 45 degrees away from the side of injection (and occasionally toward) and the X-ray tube overhead with beam projected along the base line of the skull, usually eliminates confusing bony shadows, and separates and throws into relief those elements of the circle most likely to harbor an aneurysm.

* 506 Oak Street, Cincinnati 19, Ohio.

FIG. 2. Oblique view.

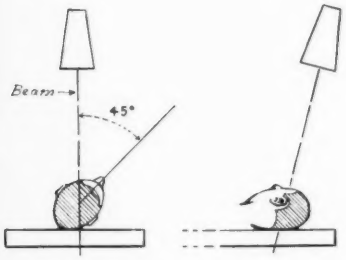


FIG. 3. Head rotated 45 degrees away from side of injection and the X-ray tube overhead with beam projected along the base line of the skull.

REFERENCE

1. KINLEY, G. J., and LEIGHNINGER, D. S. Aneurysm of anomalous ophthalmic artery presenting in the sphenoid sinus and simulating an aneurysm of the internal carotid artery on routine arteriography. *J. Neurosurg.*, 1952, *9:* 544–547.

A NEUROSURGICAL CHAIR*

W. JAMES GARDNER, M.D.

Department of Neurosurgery, The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute, Cleveland, Ohio

(Received for publication September 20, 1954)

Difficulties encountered during operations upon the head or upper spine are frequently caused by: (1) awkward positioning of the patient; (2) increased venous pressure; and (3) embarrassed respirations. In many neurosurgical procedures these factors can be minimized by placing the patient in a sitting or semireclining position in a suitable operating chair. In 1935[1] we described a neurosurgical chair and the advantages of the sitting position particularly for operations upon the cerebellum and cervical spine. Disadvantages of this position are the occasional occurrence of postural hypotension and the rare development of air embolus.

The basic requirements of a neurosurgical chair are as follows:

(1) It should maintain the body in a position that will minimize the tendency to postural hypotension and air embolism.

(2) Should hypotension develop, it must be possible to place the head lower than the feet.

(3) The chair should hold the patient's body in position without the use of restraining straps.

(4) Its shape should be such that it is impossible for the patient to slump or to struggle out of position during the course of the operation.

(5) It should have a readily adjustable headrest which will hold the head firmly, will permit the surgeon easy access to any portion of the head and upper spine, and at the same time afford the anesthetist free access to the air passages.

(6) It should be comfortable for the conscious patient.

The chair described below fulfills these requirements. The prototype of this chair originated 15 years ago and it has gradually evolved to its present form as the result of operating experience. Instead of the usual right-angled seat, this chair has a seat that is sharply flexed on the back rest, while the leg rest is extended. For the sake of stability, these angles are fixed. This shape prevents forward slumping and at the same time combats the tendency to venous pooling which contributes to postural hypotension and air embolism. To prevent sideways slumping, the back rest, seat, and leg rest are trough-shaped, which shape increases the area of contact with the patient's body and eliminates the need for padding. This in turn affords increased stability of the patient's body. Should the patient struggle at any time during operation, the force of gravity returns him to his original posture as soon as muscular contractions cease. During the 15 years that this type of chair has been used, with no padding other than a cotton blanket, no patient has had any pressure areas develop on the skin.

The important and basic feature of an operating chair is that it affords adequate

* Demonstrated at the meeting of the Harvey Cushing Society, Santa Fe, New Mexico, May 1954.

support to the skeleton. This structure is the same size in a fat as in a thin person. Chair manufacturers tend to build chairs wide and flat in order to accommodate all sizes of people, which means that they afford inadequate support. The wedging effect, which the contours of this chair furnish, permits the force of gravity to immobilize the skeletal framework without impeding respiration or circulation. With perhaps the addition of a folded blanket under the buttock or calves, a chair of this shape, though narrow, will accommodate a fat person as comfortably as a thin one and a tall person as readily as a short one. The contours of the chair

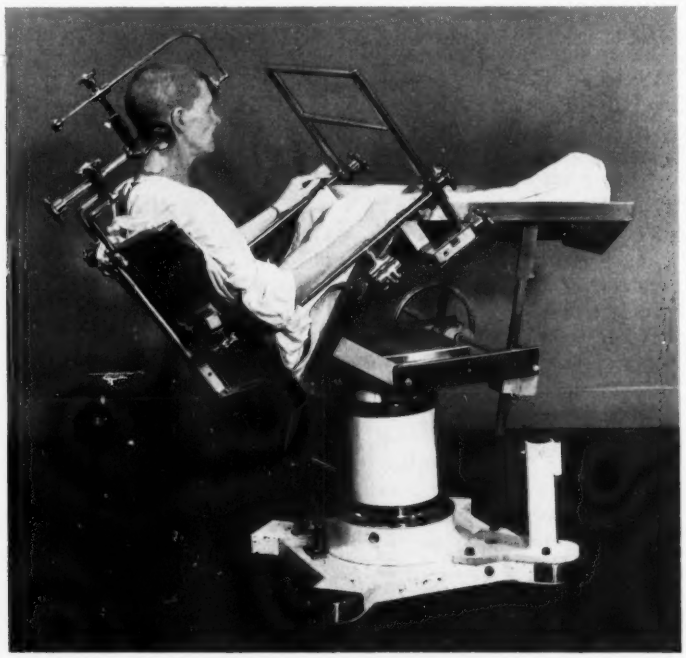


FIG. 1. Patient in position for approach to the gasserian ganglion.

enable the conscious patient to relax completely, because he feels secure and no muscular effort is required to maintain the position. This, together with the fact that the tendency to syncope is controlled, makes the chair satisfactory for use during operations on the conscious patient. The comfort of the position is attested to by the fact that during its manufacture, when the noon whistle blew, there ensued a contest among the workmen to determine who would sit in the wooden model during the lunch hour.

The chair can, of course, be raised or lowered by a foot pump, and also rotated about its vertical axis. By means of a crank, it can be tilted back like a rocking chair so that the feet are higher than the head. A slot in the back permits a lumbar puncture to be performed during operation, if indicated. Movable arm rests are provided. A small seat attaches to the back to accommodate a child.

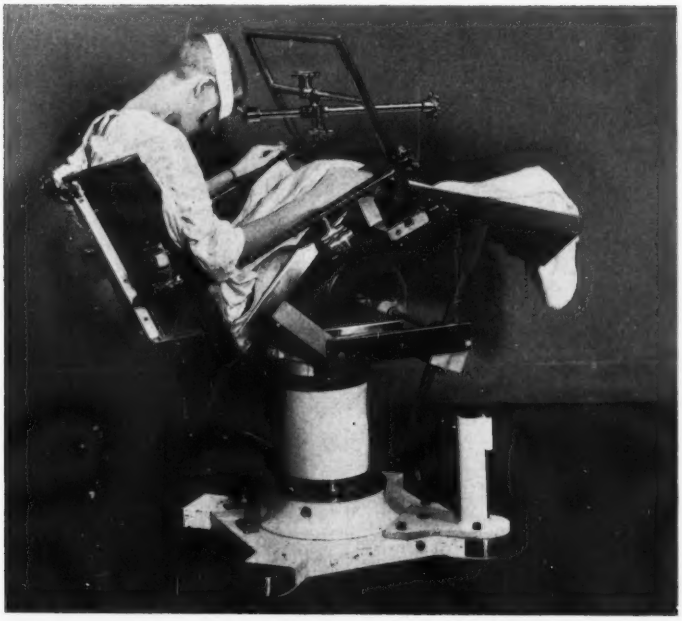


FIG. 2. Patient in position for a cerebellar operation or cervical laminectomy, demonstrating the degree of backward tilting generally employed. The socket attached to the base of the chair is to receive the patient-lifter.

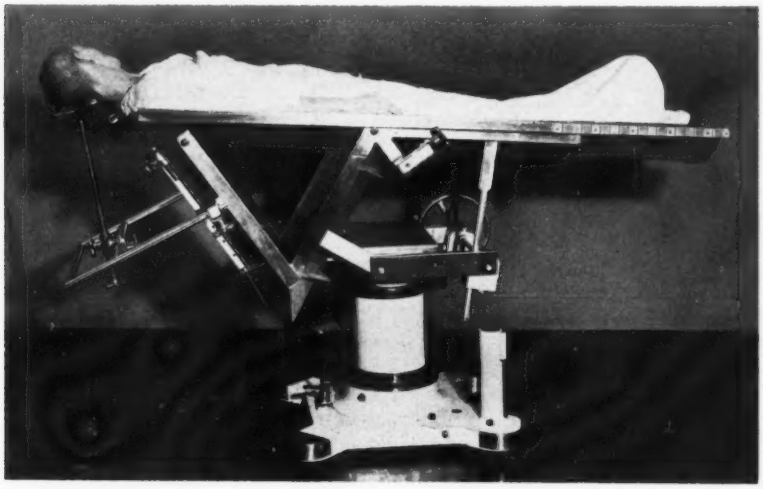


FIG. 3. The supine position for craniotomy. The table top fastens securely on the backward-tilted chair. The 3-point craniotomy headrest combines stability with ready adjustability.

The tic headrest fastens to the back of the chair. In principle, it is a three-point cushioned vise which, in the standard position, makes contact with the mastoid processes and forehead (Fig. 1). The bar to the forehead pad may be tilted to either side to afford access to a large area of the calvarium. After clamping in the tic headrest, the head may be tilted toward either shoulder if desired. When the head is placed in the clamp so that the face is toward one side, the Shelden position[2] for approach to the optic chiasm may be simulated.

In all operations in the sitting position, the chair should be tilted backward as far as feasible without interfering with the convenience of the operator. This settles the patient more firmly in position, aids venous return from the lower extremities and decreases the vertical distance between the brain and the heart. Because of the vasodepression which occasionally results from the sympathectomy effect of bilateral

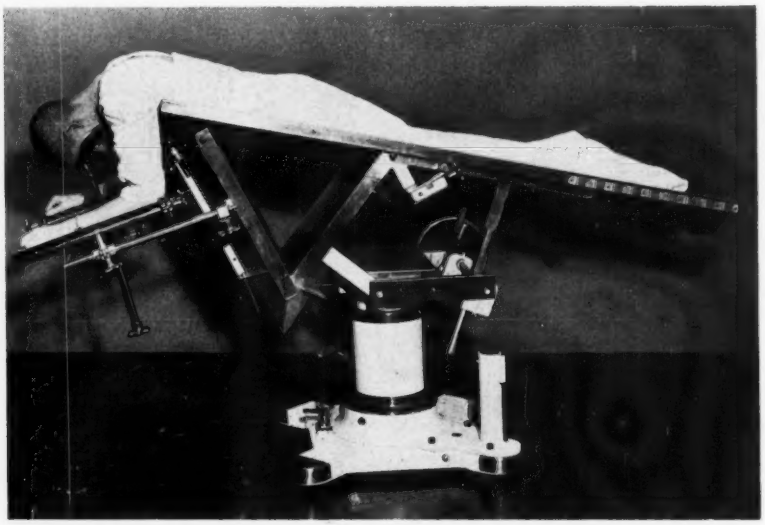


FIG. 4. The prone position for posterior craniotomy or cervical laminectomy.

cordotomy, we advocate the use of a G-suit (antigravity suit) when this procedure is carried out in the sitting position. In the Taarnhøj operation carried out in this chair, no additional precautions are ever taken to combat hypotension. A fall in blood pressure has never been observed and although the superior petrosal sinus has been opened repeatedly, aspiration of air has never happened.

The headrest pads employ a new and very effective principle. They consist of an inelastic but flexible plastic capsule, distended to the shape of a doughnut by the injection of a fluid under atmospheric pressure. This principle, by insuring an even distribution of pressure, permits a smaller sized pad to be employed and this renders a larger area of the skull accessible to the surgeon. These pads will withstand a pressure of 250 pounds without rupturing. If a pad is punctured, it may be readily replaced by a new one, although in 12 months of use this has not proved necessary. Since the contained fluid is noncompressible, it eliminates the movement inherent in

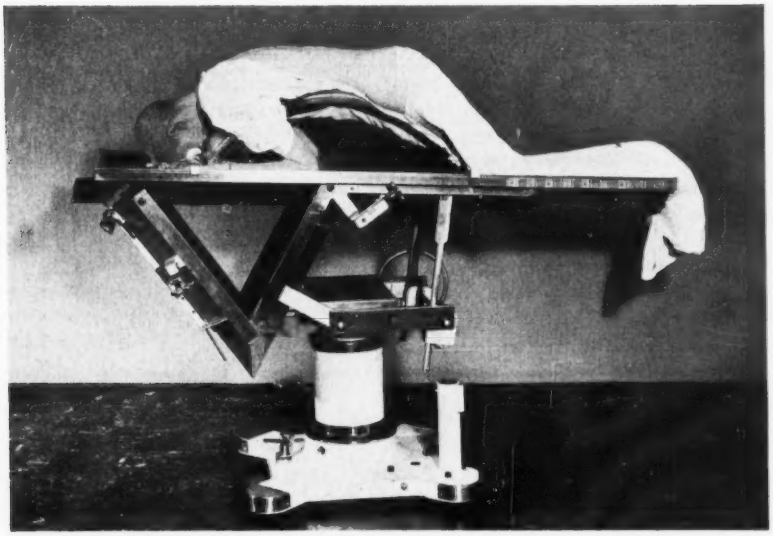


FIG. 5. The position for lumbar or lower thoracic laminectomy.

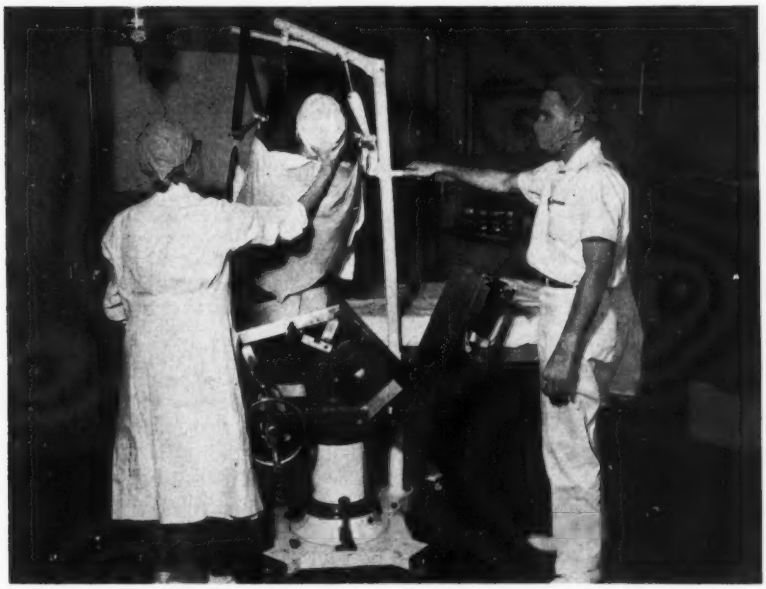


FIG. 6. The patient has been raised by the lifter and the orderly is rotating the latter in its socket preparatory to lowering the patient into the bed alongside the chair.

any compressible type of padding. The cerebellar headrest is U-shaped and designed to support the head by contact with the forehead and malar prominences (Fig. 2). Movement of the lower jaw is unimpeded and ready access is afforded to the nose and mouth. Cushioning of this rest is provided also by a water-filled capsule molded to the shape of the metal portion. The rest is attached by a locking ball-and-socket joint to a rod which, in turn, fastens, by means of a 3-way adjustable clamp, to a horizontal bar in front of the patient.

The chair is converted into an operating table by simply laying a table top across it (Fig. 3). This table top is also trough-shaped and has a slot which permits spinal puncture with the patient in the supine position. A 3-point craniotomy headrest adjusts automatically to the contour of any portion of the calvarium with the patient in the supine or lateral position. The cerebellar headrest is as effective when the patient is in the prone position as it is when he is sitting (Fig. 4). A laminectomy frame, also cushioned by fluid-filled plastic pads, fits on the table. It flexes the lumbar spine while allowing the abdomen to hang free (Fig. 5). This reduces the tendency to venous bleeding.

The removal of a patient from an operating chair has always been an awkward procedure. To overcome this difficulty, a patient-lifter has been developed. It operates as follows: A canvas sling is placed on the seat of the chair before the patient is put into position. When the operation is completed, a hydraulic hoist is fastened by hooks to each of the four corners of the canvas sling. The patient is then simply raised from the chair by means of the hoist and deposited in his bed. The hoist consists of a vertical pole to the top of which is attached a boom that can be raised or lowered by a small manually operated hydraulic pump. The pole of the hoist fits in a socket fastened to the base of the chair. After the patient has been raised from the chair, the hoist is rotated in the socket until he is over the bed which has been placed beside the chair (Fig. 6). The boom is lowered by a small release valve. The patient-lifter is detached from the chair when not in use. It can, however, be used to apply skeletal traction during operations such as the open reduction of a fracture dislocation of the cervical spine.

The author wishes to express his grateful appreciation to Mr. Valentine Seitz,* and to Mr. Emil H. Buehning of the electromechanical shop of the Cleveland Clinic for their invaluable aid in devising the various headrests and clamps. To Mr. Elmer Ries of Cincinnati goes the credit for assembling the ideas and putting the chair on a production basis.† To the Stratton Equipment Co. of Cleveland, credit is due for the development of the patient-lifter. The fluid-filled plastic pads were developed through the cooperation of the Rand Development Corporation of Cleveland and Vin Tex Sealers of Chicago.

REFERENCES

1. Gardner, W. J. Intracranial operations in the sitting position. *Ann. Surg.*, 1935, *101:* 138–145.
2. Shelden, C. H., Pudenz, R. H., and Restarski, J. S. A new position for frontal craniotomy. *J. Neurosurg.*, 1945, *2:* 546–550.

* Now deceased.

† Ries Manufacturing Co., 717 Sycamore Street, Cincinnati 2, Ohio.

NOTICES

AMERICAN BOARD OF NEUROLOGICAL SURGERY

The American Board of Neurological Surgery met in New Haven, Connecticut, Nov. 14–17, 1954. The Board is still engaged in re-evaluating residency training programs and at this meeting it was decided that a preceptorship type of training would no longer be accepted.

Examinations were given Nov. 15–17 and the following candidates were certified:

ARNOLD, ARTHUR LaGrange Medical Bldg., LaGrange, Ill.
BAIRD, WILLIAM C. 900 Second National Bldg., Akron, Ohio
BERMAN, AARON JOSEPH 470 Ocean Ave., Brooklyn 16, N. Y.
BROWNE, KENNETH M. University of Nebraska College of Medicine, Omaha, Neb.
CLARY, WARREN UPTON 228 East Huntingdon St., Savannah, Ga.
*COLCLOUGH, JOHN ASHBY 217 Carondolet Ave., New Orleans, La.
CUMMINGS, KENNETH L. 200 West Second St., Lexington, Ky.
DAVEY, LYCURGUS MICHAEL 255 Bradley St., New Haven 10, Conn.
DAVIS, COURTLAND HARWELL, JR. Bowman Gray School of Medicine, Winston-Salem 7, N. C.
EHLERT, ALBERT J. 1206 Citizens Bldg., Tampa, Fla.
ESPEY, FRANK F. 123 Mallard St., Greenville, S. C.
FAIRMAN, DAVID Juncal 1360, Buenos Aires, Argentina
HADDAD, FAUD SAMI Orient Hospital, Beirut, Lebanon
HUNT, WILLIAM E. 9 Buttles Ave., Columbus 8, Ohio
KELL, JOSEPH FOSTER, JR. 1200 East Broad St., Richmond 19, Va.
KING, ROBERT BAINTON 6227 Rosebury Ave., St. Louis 5, Mo.
LLEWELLYN, RAEBURN C. 3503 Prytania St., New Orleans, La.
LUROS, JOHN THEODORE 2901 North Meridian, Indianapolis, Ind.
MARTIN, LUTHER C. 82 Rutledge Ave., Charleston, S. C.
MENSE, JACKSON S. 117 East Eighth St., Long Beach 13, Calif.
RAND, ROBERT WHEELER University of California Medical Center, Los Angeles 24, Calif.
REED, H. LESTER 315 Fincastle Bldg., Louisville 2, Ky.
ROLLINS, STACY LINTON, JR. 1150 Connecticut Ave., N.W., Washington, D. C.
ROSEGAY, HAROLD USAH Neubrucke, A.P.O. 34, New York, N. Y.
SCHRICKER, JAMES LOUIS, JR. Fitzsimmons Army Hospital, Denver, Colo.
SKULTETY, F. MILES Dept. of Surgery, University Hospitals, Iowa City, Iowa
SLADE, HARRY W. 3395 Scranton Road, Cleveland 9, Ohio
SMITH, GEORGE WILLIAMS Johns Hopkins Hospital, Baltimore, Md.
THELEN, EMIL P. U. S. Naval Hospital, Bethesda, Md.
VAN ALLEN, MAURICE WRIGHT Veterans Administration Hospital, Iowa City, Iowa
WEADON, PRESTON STENZ 211 Bronson Medical Center, 252 East Lovell St., Kalamazoo, Mich.
WETZEL, NICHOLAS 700 North Michigan Ave., Chicago 11, Ill.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

At the 34th annual meeting of the Association for Research in Nervous and Mental Disease held in New York City on December 10 and 11, 1954, the following officers were elected for the year 1955: President, Dr. J. E. Moore; 1st Vice-President, Dr. John Romano; 2nd Vice-President, Dr. David Seegal; Secretary-Treasurer, Dr. Clarence C. Hare; Assistant Secretary, Dr. Rollo J. Masselink.

The subject for the 1955 meeting will be "The Neurological and Psychiatric Aspects of the Disorders of Aging." The meeting will be held on December 9 and 10, 1955 in New York City.

* Certified in May 1954 in Chicago, Illinois.

SOCIETY OF NEUROLOGICAL SURGEONS

The next annual meeting of the Society of Neurological Surgeons is to be held in Chicago on April 25 and 26, 1955. The host will be Dr. Loyal Davis, and the headquarters will be at the Drake Hotel. Attendance is for the members and invited guests.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Conn., U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbreviations of names of periodicals should conform to the style used in *A world list of scientific periodicals published in the years 1900–1933* (Oxford University Press, 1934).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

ILLUSTRATIONS. Care should be taken not to use too many illustrations, since the Journal provides no figures free of charge. Color plates also are inserted at the expense of the contributor. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead.

SPECIAL TYPOGRAPHY. Tables and also text set up in small print in excess of 10 per cent of composition costs are charged to the author.

PROOF CORRECTIONS will be allowed without charge up to 5 per cent of the original cost of composition. The cost of changes in page proof, if made by the author, will be borne by him. *Page proof* will be submitted to authors only when special questions arise.

REPRINTS. Owing to existing paper shortage, no reprints may be supplied *gratis*. A schedule of charges for reprints is noted on the order forms accompanying the galley proof of each paper.

SUBSCRIPTIONS. The subscription rate for the volume of six bi-monthly issues is $11.00 in the United States and Pan-American Union; $11.50 in Canada; $12.00 in other countries. Single copies are $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: A Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available. Volume I, Number 1 is supplied only on orders for the complete volume. Payments may be made in advance to Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

CONTENTS

1. Aneurysms of the Middle Cerebral Artery. Report of Seven Operative Cases; Review of Literature; Evaluation of Surgical Therapy. DANIEL PETIT-DUTAILLIS, and HAL W. PITTMAN 1

2. Decerebrate State in Children and Adolescents. ROBERT C. L. ROBERTSON, and CLAUDE POLLARD, JR. 13

3. Some Efferent Connexions of the Human Frontal Lobe. ALFONSO MARTÍNEZ 18

4. Occlusion of the Middle Cerebral Artery Under Normotension, and Anemically Induced and Chemically Induced Hypotension. BRUCE RALSTON, THEODORE RASMUSSEN, and THELMA KENNEDY 26

5. Intracranial Hematomas Associated with Penetrating Wounds of the Brain. JOSEPH C. BARNETT, and ARNOLD M. MEIROWSKY .. 34

6. An Electrophysiological Study of Nerve Regeneration in the Cat. WILLIAM F. COLLINS, JAMES L. O'LEARY, WILLIAM E. HUNT, and HENRY G. SCHWARTZ 39

CASE REPORTS AND TECHNICAL NOTES:

A Case of Persistent Carotid-Basilar Anastomosis Associated with Aneurysm of the Homolateral Middle Cerebral Artery Manifested by Oculomotor Palsy. FREDERICK MURTAGH, HERBERT M. STAUFFER, and ROBISON D. HARLEY 46

Pterional Meningioma "En Plaque." Report of a Case of Thirty-Six Years' Duration. KENNETH H. ABBOTT, and BERNARD GLASS ... 50

Solitary *Aspergillus* Granuloma of the Brain. Report of 2 Cases. IRA J. JACKSON, KENNETH EARLE, and JOSE KURI 53

A Case of Carotid-Basilar Anastomosis with Multiple Associated Cerebrovascular Anomalies. J. P. SCHAERER 62

Arachnoidal Cysts of the Cerebellopontine Angle. FRANCISCO GARCÍA BENGOCHEA, and FRANCISCO LEÓN BLANCO 66

Osteochondroma of the Lumbar Spine. Report of a Case. HUSAMEDDIN GOKAY, and PAUL C. BUCY 72

The Oblique View in Cerebral Angiography. CURWOOD R. HUNTER, and FRANK H. MAYFIELD 79

A Neurosurgical Chair. W. JAMES GARDNER 81

NOTICES 87

GEORGE BANTA PUBLISHING COMPANY, MENASHA, WISCONSIN